
**Cognitive, Interpersonal and Psychosocial Factors Influencing
Vulnerability, Treatment Outcome and Relapse in
Bipolar Affective Disorders**

A Clinical Randomised Controlled Treatment Trial

Matthias Schwannauer



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Contents

ABSTRACT	I
I INTRODUCTION.....	3
II BIPOLAR DISORDERS	II
2.1 CONCEPT AND PHENOMENOLOGY	11
2.2 PSYCHOPATHOLOGY AND ASSESSMENT	12
2.3 DIAGNOSIS AND DEFINITIONS OF BIPOLAR DISORDER.....	14
2.4 ONSET AND COURSE OF THE DISORDER.....	17
2.5 PSYCHOSOCIAL FACTORS.....	18
2.5.1 Life events and difficulties.....	20
2.5.2 Childhood Stressful Life Events	35
2.6 THE INTERPERSONAL CONTEXT.....	40
2.6.1 Social Networks and Social Support.....	40
2.6.2 Conceptualisation of Social Support.....	42
2.6.3 Areas and Resources of Social Support	46
2.6.4 Models of Social Support.....	48
2.6.5 Positive and negative aspects of social support.....	54
2.6.6 Social Support and Psychological Disorders.....	58
2.6.7 Moderating influences of Social Support.....	59
2.6.8 Family factors.....	69
2.6.9 Expressed Emotion	70
2.6.10 Operationalisation of Expressed Emotion	72
2.6.11 Expressed Emotion and Psychological Health.....	77
2.6.12 Psycho-social intervention based around the EE concept.....	82
2.6.13 Social Support and Expressed Emotion	90
2.7 PSYCHOLOGICAL FACTORS	95
2.7.1 Dysfunctional Attitudes.....	95
2.7.2 Self esteem / Self concept.....	100
2.7.3 Affect and emotion related personality traits.....	103
2.7.4 Impulsivity	107
2.7.5 Affect Regulation.....	107
2.8 PSYCHOLOGICAL MODELS OF BIPOLAR DISORDER.....	110
2.8.1 Model of behavioural activation and behavioural inhibition.....	110
2.8.2 Circadian and social rhythms in bipolar disorder.....	113
2.8.3 A CBT model of Bipolar Affective Disorder.....	117
2.9 SUMMARY	122
III PSYCHOLOGICAL INTERVENTIONS FOR BIPOLAR DISORDERS	127
3.1 SCOPE FOR PSYCHOLOGICAL INTERVENTION & PREVENTATIVE STRATEGIES	127
3.2 ASSESSMENT & PSYCHOLOGICAL FORMULATION.....	128
3.2.1 Psychoeducational approaches	128
3.2.2 Cognitive therapy approaches.....	136
3.2.3 Interpersonal approaches.....	151
3.2.4 Family Therapy approaches.....	157
3.2.5 Long term and psychiatric management issues	166
3.3 SUMMARY	170

IV A COGNITIVE INTERPERSONAL APPROACH TO RECOVERY AND RELAPSE PREVENTION.....	188
4.1 CBT FOR BIPOLAR DISORDERS.....	188
4.1.1 Self monitoring and prodromal changes.....	190
4.1.2 Cognitive strategies.....	193
4.1.3 Interpersonal functioning.....	200
4.2 PSYCHOLOGICAL FACTORS IN VULNERABILITY TO RELAPSE.....	201
4.2.1 Affect, meaning and relapse.....	202
4.2.2 A cognitive behavioural model of early signs and relapse.....	206
4.2.3 Trauma.....	213
4.2.4 Interpersonal coping.....	215
4.2.5 Individual adaptation to onset of bipolar disorder.....	219
4.3 SUMMARY.....	222
V PREDICTORS OF SYMPTOM SEVERITY AND TREATMENT OUTCOMES IN BIPOLAR DISORDERS – A RANDOMISED CONTROLLED TREATMENT TRIAL.....	224
5.1 INTRODUCTION.....	224
5.2 AIMS & HYPOTHESES.....	231
5.2.1 Hypotheses.....	232
5.3 STUDY DESIGN.....	233
5.3.1 Power.....	235
5.4 ETHICAL APPROVAL.....	236
5.5 PSYCHOLOGICAL TREATMENT.....	237
5.6 SAMPLE.....	239
5.7 METHODS.....	243
5.7.1 Assessment & Measures.....	243
5.8 STATISTICAL ANALYSIS.....	260
VI TREATMENT OUTCOMES.....	262
6.1 SAMPLE CHARACTERISTICS AT BASELINE.....	262
6.1.1 Demographic Characteristics.....	262
6.1.2 Demographic Psychosocial Characteristics.....	263
6.1.3 Clinical Characteristics.....	264
6.2 STATISTICAL ANALYSIS.....	267
6.2.1 Assumptions.....	268
6.3 TRIAL OUTCOME – INTENT TO TREAT ANALYSIS.....	268
6.3.1 Main treatment effect for primary outcome – Quality of Life.....	269
6.3.2 Main treatment effects for secondary outcomes – Observer rated measures of symptom severity.....	272
6.3.3 Main treatment effects for secondary outcomes – Self-report measures of symptom severity.....	276
6.3.4 Gender effects on main treatment effect.....	278
6.4 OUTCOME INDICATORS FOLLOWING PSYCHOLOGICAL INTERVENTION.....	280
6.4.1 Outcomes – Quality of Life.....	280
6.4.2 Outcomes – Observer rated measures of symptom severity.....	284
6.4.3 Outcomes – Self report measures of symptom severity.....	288
6.4.4 Relapse and recurrence.....	294
6.5 EFFECT OF DELAYED TREATMENT CONDITION ON TREATMENT EFFECTS OVER TIME.....	296
6.6 SUMMARY.....	298

VII COGNITIVE, INTERPERSONAL AND PSYCHOSOCIAL PREDICTORS OF TREATMENT EFFECTS.....	301
7.1 PSYCHOLOGICAL AND PSYCHOSOCIAL SAMPLE CHARACTERISTICS AT BASELINE	302
7.1.1 Cognitive and psychological variables	302
7.1.2 Psychosocial variables.....	304
7.2 CHANGES IN PSYCHOLOGICAL AND PSYCHOSOCIAL CHARACTERISTICS FOLLOWING TREATMENT	305
7.2.1 Psychological variables	305
7.2.2 Psychosocial variables.....	308
7.3 STATISTICAL ANALYSIS	309
7.4 BASELINE PREDICTOR MODELS.....	311
7.4.1 Predictors of bipolar symptoms at baseline.....	311
7.4.2 Predictors of Quality of Life at baseline	313
7.5 PREDICTORS OF OUTCOME.....	315
7.5.1 Predictors of bipolar symptoms following treatment.....	317
7.5.2 Predictors of quality of life following treatment.....	318
7.5.3 Predictors of relapse and recurrence	320
7.6 SUMMARY	323
VIII STRUCTURAL MODELS OF TREATMENT EFFECTS.....	327
8.1 HYPOTHESES	328
8.2 STATISTICAL ANALYSIS	330
8.2.1 Methodological assumptions.....	332
8.3 EFFECTS OF PREDICTORS OF BIPOLAR SYMPTOMS AT BASELINE.....	335
8.3.1 Predictor model of general psychiatric symptomatology at baseline.....	335
8.3.2 Predictor model of depression at baseline	336
8.3.3 Predictor model of mania at baseline.....	339
8.4 PREDICTORS OF OUTCOME.....	341
8.4.1 Predictor model of general psychiatric symptomatology following treatment.....	341
8.4.2 Predictor model of depression following treatment.....	342
8.4.3 Predictor model of mania following treatment	344
8.5 SUMMARY	346
IX DISCUSSION AND INTEGRATION OF FINDINGS.....	351
9.1 TRIAL DESIGN AND IMPLEMENTATION.....	352
9.1.1 Patient preference	353
9.1.2 Retention and involvement of participants	356
9.2 KEY OUTCOMES OF COGNITIVE INTERPERSONAL THERAPY IN BIPOLAR DISORDER.....	358
9.3 PSYCHOLOGICAL MODELS OF BIPOLAR DISORDER.....	361
9.3.1 Predictors of bipolar symptoms	362
9.3.2 Integration of cognitive and interpersonal factors in bipolar disorder	365
9.4 THEORETICAL IMPLICATIONS.....	370
9.5 LIMITATIONS OF THE PRESENT STUDY	372
9.7 FUTURE RESEARCH	374
X PAPERS ORIGINATING FROM THE PRESENT STUDY	376
XI REFERENCES	376
APPENDIX.....	437
SEMI-STRUCTURED INTERVIEW GUIDE AND ASSESSMENT TOOLS.....	437

Tables and Figures

TABLE 2.1: CLASSIFICATION OF BIPOLAR AFFECTIVE DISORDER	15
TABLE 2.2: MAIN ASPECTS OF SOCIAL SUPPORT.....	48
TABLE 2.3: STUDIES INTO VARIOUS BEHAVIOUR AND ATTITUDE CHARACTERISTICS OF FAMILIES WITH HIGH LEVELS OF EE	75
TABLE 3.1: CBT AND IPSRT OUTCOME STUDIES	178
FIGURE 5.1: PARTICIPANT FLOW	242
TABLE 6.1: BASELINE CHARACTERISTICS OF THE SAMPLE BY TREATMENT GROUP	262
TABLE 6.2: BASELINE SOCIAL NETWORK CHARACTERISTICS BY TREATMENT GROUP	264
TABLE 6.3: HISTORY OF ILLNESS AND SERVICE USE BY TREATMENT GROUP.....	265
TABLE 6.4: CLINICAL CHARACTERISTICS BY TREATMENT GROUP	266
TABLE 6.5: PSYCHOLOGICAL TREATMENT & TAU VERSUS TAU – QUALITY OF LIFE.....	270
FIGURE 6.1: TIME X GROUP – QUALITY OF LIFE	272
TABLE 6.6: PSYCHOLOGICAL TREATMENT & TAU VERSUS TAU – OBSERVER RATED MEASURES OF PSYCHIATRIC SYMPTOMS	274
FIGURE 6.2: TIME X GROUP – OBSERVER RATED PSYCHIATRIC SYMPTOMS.....	275
TABLE 6.7: PSYCHOLOGICAL TREATMENT & TAU VERSUS TAU – OBSERVER RATED MEASURES OF PSYCHIATRIC SYMPTOMS	277
FIGURE 6.3: TIME X GROUP – BDI-II	277
FIGURE 6.4: TIME X GROUP – BLIP MANIA AND BLIP DEPRESSION.....	278
TABLE 6.8: REPEATED MEASURES ANOVA'S FOR QUALITY OF LIFE (WHOQOL-BREF)	280
TABLE 6.9: PAIRWISE COMPARISONS OF WHOQOL-BREF DOMAIN 1 OVER TIME.....	281
TABLE 6.10: PAIRWISE COMPARISONS OF WHOQOL-BREF DOMAIN 2 OVER TIME.....	281
TABLE 6.11: PAIRWISE COMPARISONS OF WHOQOL-BREF DOMAIN 3 OVER TIME.....	282
TABLE 6.12: PAIRWISE COMPARISONS OF WHOQOL-BREF DOMAIN 4 OVER TIME.....	282
FIGURE 6.5: QUALITY OF LIFE SCORES OVER TIME – PSYCHOLOGICAL WELL BEING	283
FIGURE 6.6: QUALITY OF LIFE SCORES OVER TIME – SOCIAL RELATIONSHIPS	284
TABLE 6.13: REPEATED MEASURES ANOVA'S FOR OBSERVER RATED MEASURES OF SYMPTOM SEVERITY	285
TABLE 6.14: PAIRWISE COMPARISONS OF PANSS GENERAL SCORE OVER TIME POINTS.....	286
TABLE 6.15: PAIRWISE COMPARISONS OF BPRS GENERAL SCORE OVER TIME POINTS.....	286
FIGURE 6.7: PANSS SCORES OVER TIME.....	287
FIGURE 6.8: BPRS SCORES OVER TIME.....	288
TABLE 6.16: REPEATED MEASURES ANOVA'S FOR SELF REPORT MEASURES OF SYMPTOM SEVERITY AND QUALITY OF LIFE	289
TABLE 6.17: PAIRWISE COMPARISONS OF BDI-II OVER TIME POINTS.....	290
FIGURE 6.9: BDI SCORES OVER TIME.....	290
TABLE 6.18: PAIRWISE COMPARISONS OF BLIP MANIA OVER TIME POINTS	291
FIGURE 6.10: BLIP MANIA SCORES OVER TIME	292
TABLE 6.19: PAIRWISE COMPARISONS OF BLIP DEPRESSION OVER TIME POINTS	292
FIGURE 6.11: BLIP DEPRESSION SCORES OVER TIME	293
TABLE 6.20: PAIRWISE COMPARISONS OF BLIP MIXED SYMPTOMS OVER TIME.....	293
FIGURE 6.12: BLIP MIXED SYMPTOMS SCORES OVER TIME.....	294
TABLE 6.21: HOSPITAL ADMISSIONS AND SERVICE CONTACTS PRE AND POST INTERVENTION	295
TABLE 7.1: PSYCHOLOGICAL CHARACTERISTICS BY TREATMENT GROUP	303
TABLE 7.2: PSYCHOSOCIAL CHARACTERISTICS BY TREATMENT GROUP	304
TABLE 7.3: REPEATED MEASURES ANOVA'S FOR PSYCHOLOGICAL VARIABLES.....	306
FIGURE 7.1: CHANGE IN PREDICTOR VARIABLES OVER TIME.....	307
TABLE 7.4: REPEATED MEASURES ANOVA'S FOR PSYCHOSOCIAL VARIABLES	308
FIGURE 7.2 CHANGES IN PERCEPTION OF SOCIAL SUPPORT	308
FIGURE 7.3 CHANGES IN OVERALL SOCIAL SUPPORT	309
TABLE 7.5: FACTOR LOADINGS ON THREE DIMENSIONS OF BIPOLAR SYMPTOMS	311
TABLE 7.6: PEARSON'S CORRELATIONS OF DISTRESS DIMENSIONS	311

TABLE 7.8: MULTIPLE REGRESSION ANALYSES AND PREDICTORS FOR SYMPTOM DIMENSIONS	312
TABLE 7.9: MULTIPLE REGRESSION ANALYSES AND PREDICTORS FOR QUALITY OF LIFE (WHOQOL-BREF) – AT BASELINE.....	314
TABLE 7.10: FACTOR LOADINGS ON THREE DIMENSIONS OF BIPOLAR SYMPTOMS AT END OF TREATMENT.....	316
TABLE 7.11: FACTOR LOADINGS ON THREE DIMENSIONS OF BIPOLAR SYMPTOMS AT FOLLOW UP.....	316
TABLE 7.12: MULTIPLE REGRESSION ANALYSES AND PREDICTORS FOR BIPOLAR SYMPTOM DIMENSIONS AT END OF TREATMENT	317
TABLE 7.13: MULTIPLE REGRESSION ANALYSES AND PREDICTORS FOR QUALITY OF LIFE (WHOQOL-BREF) – AT OUTCOME	319
TABLE 7.14: LOGISTIC REGRESSION ANALYSES AND PREDICTORS FOR RELAPSE AT 18 MONTH FOLLOW UP.....	321
FIGURE 8.1: SCHEMATIC BASELINE MEDIATION MODEL FOR DEPRESSION AND MANIA	329
FIGURE 8.2: SCHEMATIC MODEL OF DIRECT AND MEDIATION EFFECTS FOR KEY OUTCOME VARIABLES .	330
FIGURE 8.3: SEM OF DEPRESSION AT BASELINE	338
FIGURE 8.4: SEM OF MANIC SYMPTOMS AT BASELINE	340
FIGURE 8.5: SEM OF DEPRESSION FOLLOWING TREATMENT	343
FIGURE 8.6: SEM OF MANIC SYMPTOMS FOLLOWING TREATMENT	345

Abstract

Bipolar Affective Disorder is one of the most long-term recurrent mental health disorders. Despite the efforts in pharmacological management of bipolar disorder, relapse and residual symptoms remain a major factor in the development of illness chronicity, and long term social and occupational disability. For individuals themselves relapse is critical in the development of secondary psychological morbidity.

On the basis of a review of the current literature on psychological treatments and the influence of psychological and psychosocial factors on the onset, course and outcome in bipolar disorder an integrated psychological intervention model for this clinical group is presented. This treatment approach will combine effective cognitive behavioural therapy elements and interpersonal psychotherapy components.

The efficacy and effectiveness of this intervention is investigated through a clinical randomised controlled trial. The trial is comparing three conditions in an exploratory partially randomised design; a waiting list condition of treatment as usual (TAU), consisting of clinical management and psychiatric follow up, and the experimental groups of cognitive interpersonal intervention in a group and individual treatment format. Participants were randomised into the TAU or treatment group; in the treatment group participants' were offered a choice of group or individual therapy. Participants randomised to TAU were offered treatment after a six months waiting period.

Those participants in the treatment group were assessed at the start of treatment, at mid-treatment, end of treatment, and at a six months and 18 months follow up using a variety of clinician rated and self rated assessments of clinical symptoms as well as relevant psychological and psychosocial factors. Clinical service data relating to service use and hospital admissions were collected for the entire group for pre and post intervention periods. Overall out of 258 referrals, 212 individuals were assessed for the study and 193 individuals started treatment, 174 participants completed the minimum number of treatment sessions, 134 were available for follow-up assessments at 6 months post treatment and 108 were available for follow-up at 18 months post treatment.

The direct comparison of treatment and control group showed a large positive treatment effect of cognitive interpersonal therapy on the primary outcome, quality of life. Similarly medium to large treatment effects were shown for the secondary outcomes, indicators of bipolar symptoms, emotional distress and indicators of relapse and recurrence.

Participants who completed treatment showed significant improvement in quality of life, psychiatric symptoms, and emotional distress'. Further, their relapse rates and hospital admissions as well as their use of emergency psychiatric services were significantly reduced. The analysis of psychological and psychosocial predictors established clear differential effects of psychological factors on therapeutic change and outcome in relation to depression and symptoms of mania respectively, demonstrating that the change in cognitive, interpersonal and psychosocial variables through the intervention is predictive of outcome indicators at end of treatment and follow up. The results of these analyses aid the development of a cognitive interpersonal model of bipolar affective disorder and support the development of an integrated psychological treatment aimed at this complex and chronic clinical group.

I Introduction

Bipolar disorder affects between 0.8% and 1.6% of the population (e.g. Kessler, et al., 1994, 1997). The mean onset is located in late adolescence and early adulthood which causes lasting psychosocial difficulties partly due to the impact of the age of onset and the crucial impact on individual development (Ramana & Bebbington, 1995), but also as a result of the high likelihood of repeated episodes within few years in 80% to 90% of the bipolar population (Goodwin & Jamison, 1990). One of the tragic manifestations of the complexity and the lasting impairments often caused by the traumatic impact of early and multiple episodes is the high suicide rate in bipolar disorder.

The disorder is identified and characterised primarily by shifts in polarity of mood, from depressed to manic. It is these shifts which differentiate bipolar from unipolar disorders, and are crucial in its diagnosis (Bebbington & Ramana, 1995). The shifts vary in severity, length and expression from episode to episode and individual to individual. For the majority of patients the illness appears to start with episodes of depression and the illness is predominantly characterised by episodes of the same polarity as the first one (Perugi et al., 2000).

Bipolar disorder encompasses four defined abnormal mood states: depression; mania; hypomania; and mixed episode. These mood states can surface at varying points in the course of the disorder, and each may dictate a different therapeutic intervention. Furthermore the period of transition between these states may also considered be

considered a 'state' in itself, and is worthy of intervention too. The complexity of the disorder presents great challenges to treatment researchers (Sachs et al., 2003).

The clinical manifestations of bipolar disorder are exceptionally diverse (Muller-Oerlinghausen et al., 2002). They range from mild hypomania or mild depression to severe forms of mania or depression accompanied by profound psychosis. The heterogeneity within the bipolar spectrum is reflected in the large variation of related research findings.

Within the spectrum, bipolar disorder can be subdivided into bipolar I disorder, a recurrent mood disorder, featuring either one or more manic and/or mixed episodes and at least one major depressive episode, or bipolar II disorder, characterised by one or more episodes of major depression and at least one hypomanic episode.

It is a disorder which affects males and females in equal proportions (Kessler et al, 1993; Kawa, 2005) with the exception of rapid cycling, a severe variant of the disorder affecting 10-15% of bipolar patients, which arises mostly in women (Calabrese et al., 2001; Muller-Oerlinghausen et al., 2002). Individual experience of bipolar varies between the sexes (Kessing, 2004). A greater incidence of depression has been found in women (Christensen et al., 2003) and they are more likely to present depressed at their first episode (Viguera et al., 2001; Kawa et al., 2005). Males are more likely to be younger at first episode (Kennedy et al., 2005). Gender differences have also been observed in comorbid disorders in patients with bipolar disorder. A higher frequency of panic and eating disorders has been found in women (McElroy et al., 2001) whereas men are more prone to substance abuse (Kessing, 2004) and obsessive-compulsive disorder (Benedetti et al., 2007).

The peak age of onset of bipolar disorder falls between 15 and 24 years (Muller-Oerlinghausen et al., 2002), although there is often up to a 10 year interval before treatment is obtained. This period is often referred to as the duration of untreated illness. The diagnosis of bipolar disorder in pre-pubertal populations remains difficult and often controversial (Kyte et al., 2006) despite reports which suggest that up to two-thirds of adult patients began having symptoms in their childhood or adolescence (Lish et al., 1994; Perlis et al., 2004). Compared with adult onsets, clinical studies have described childhood-onset bipolar disorder as characterised by increased presence of psychosis, poorer outcome, longer duration of illness, slower recovery times, more recurrences, greater comorbidity with other disorders, and a greater incidence of suicide (for review see Kyte et al. 2006). In terms of psychosocial functioning, adolescents with paediatric bipolar disorder report lower self-esteem, more hopelessness, more traumatic experiences and negative life events, greater difficulties in regulating emotion, and poorer coping strategies than adolescent controls (Rucklidge, 2006).

Various clinical and epidemiological studies have documented high rates of comorbid disorders among individuals with bipolar disorder. The disorder is associated with a significant risk of substance abuse (Thase & Salloun, 2000), high rates of anxiety disorder (McElroy et al., 2001; Otto et al., 2006), personality disorder (Barbato & Hafner, 1998; Brieger et al., 2003), and to a lesser extent eating disorders (McElroy et al., 2001). In an epidemiological study of bipolar outpatients, McElroy et al. (2001) found at least 65% of their sample met criteria for at least one comorbid axis I disorder. Furthermore, comorbidity was associated with an earlier age of onset and greater illness severity. Other studies show comorbid conditions to be associated with poorer response to treatment (Henry et al., 2003; Schmitz et al., 2006) and an increased risk of suicide

attempt (Simon et al., 2007), thus highlighting the need for specific interventions targeting these groups.

Psychotic symptoms are commonly observed in bipolar disorder, affecting approximately 75% of manic patients (Tohen et al., 1990). Studies have shown that patients with a prior history of psychotic symptoms showed poorer outcome and a more severe course of illness (Tohen et al., 1990; Miklowitz, 1992).

Bipolar disorder is also associated with significant risk of suicide attempt (see Hawton et al., 2005 for review). Up to around 50% of all individuals with bipolar disorder will make a suicide attempt (Valtonen et al., 2005). As may be expected, both attempts and ideation are associated with depressive aspects of the illness, and hopelessness and severity of depression are key indicators of risk (Valtonen et al., 2007). Despite being a largely state-dependent phenomenon, there is a need for focus on suicidality not only during acute episodes but also during preventative and maintenance care due to the progressive, episodic, and chronic nature of bipolar depression (Rucci et al., 2002).

The course of illness is highly variable, but is characterised by frequent relapse (Keller et al., 1982; Goodwin & Jamison, 1990; Gitlin et al., 1995). Around half of individuals relapse requiring hospitalisation within three to four years (Goldberg et al., 1995; Kessing et al., 2004a; Bromet et al., 2005). Unfortunately with each relapse the risk of subsequent relapse increases (Keller et al., 1982; Kessing et al., 2004a) and the time between episodes often shortens as duration of illness lengthens (Goodwin & Jamison, 1990). Relapse occurs due to various factors such as stressful life-events, non-compliance of medication, dose reduction, or the natural progression of the illness (Leverich et al., 1990). Predictors of relapse include stressful life events, increased number of previous

episodes, shorter time between episodes, persistence of affective symptoms and episodes (Altman et al., 2006).

Even when individuals are euthymic they appear unlikely to be completely symptom free. Inter-episode sub-syndromal symptoms can be easy to miss (Morriss, 2002), but are three times more likely to occur than syndromal symptoms (Scott et al., 2000; Judd et al., 2002). Paykel et al. (2006) found that sub-syndromal symptoms are present twice as long as major symptoms, thus constituting a considerable clinical element over time. Observational studies confirm that inter-episode symptoms are related to impaired function and reduced time to relapse (Perlis et al., 2006), and residual depressive symptoms are negatively correlated with quality of life (Gazalle et al., 2006). There is further a clear effect of the disorder's phenomenology in that individuals are much more likely to have sub-syndromal depression than mania.

Psychosocial and psychological impairments following recovery from a severe episode are evident for a number of years (Coryell et al, 1993; Goldberg & Harrow, 2004; Gitlin et al, 1995). An array of studies now exist showing reductions in psychosocial functioning in bipolar disorder, with deficits found in social functioning, particularly family relationships (for review see Elgie et al., 2007) and employment functioning (e.g. Waghorn et al., 2007). In their 2001 review, MacQueen et al. found that 30-60% of individuals with bipolar disorder fail to regain full occupational and social functioning. Ameen & Ram (2007) concur that negative symptoms are prevalent in remitted bipolar disorder and contribute to patients' socio-occupational dysfunction. Following recurrence, admission to hospital, work impairment, and overall poor functioning is more common in bipolar than unipolar disorder (Goldberg, Harrow & Grossman, 1995).

Research into quality of life as measuring illness intrusiveness has shown bipolar disorder to be at least as intrusive as a number of chronic medical conditions, including end stage renal disease and rheumatoid arthritis (Robb et al, 1997). This impairment in quality of life was also found in patients who were euthymic. Akvardar et al. (2006) found that the quality of life of a sample of Turkish psychiatric patients (including bipolar patients) was worse than in patients with diabetes.

It has long been observed that patients with bipolar disorder suffer from cognitive impairment. There is a broad consensus that euthymic bipolar patients perform worse on neuropsychological tests of attention, verbal and non-verbal memory, and executive function compared to healthy controls (Dittmann et al., 2007). Recently, there has been increasing recognition that these deficits do not appear to be state-dependent and are apparent during periods of euthymia (Martínez-Arán et al., 2004a). This impairment continues to be evident when sub-syndromal symptoms are controlled for. Recent studies, particularly the work of Martínez-Arán and various colleagues (2002, 2004a,b, 2007) have shown the impact of cognitive dysfunction on psychosocial functioning in bipolar patients. In particular they have shown certain measures e.g. verbal memory can predict psychosocial functioning (Martínez-Arán et al., 2007). Zubieta et al. (2001) found social and occupational functioning was associated with certain cognitive deficits, as well as number of episodes of depression and mania.

One of the main challenges facing the psychological therapist in bipolar disorder is the strong heterogeneity of this disorder group and its various phenomenological manifestations. In comparison with other mood disorders the emotional, cognitive and behavioural problems associated with bipolar disorder range from long periods of

depression to varying degrees of euphoria, irritability, agitation and psychotic symptomatology. Most individuals suffering from bipolar disorder experience cyclical symptoms and multiple episodes of both depression and mania over the life-span which creates significant disruptions in the patients' lives as well as lasting psychological and psychosocial difficulties.

A second challenge for the treatment of bipolar disorder is the high proportion of comorbidity. Clinically significant are the high prevalence rates of substance abuse, up to 61% (Brady & Lydiard, 1992), a large proportion of 21% of individuals suffering from a bipolar disorder also suffer from an anxiety disorder (Himmelhoch, 1999), and 50% display difficulties associated with personality disorders (Peselow, et al., 1995). These high rates of comorbidity create clinical complexity not only in the assessment of current difficulties and realistic treatment goals, but also in terms of the cognitive behavioural treatment of core symptoms and psychosocial impairments, as these difficulties are often masked or confused by heightened depressive or manic symptoms.

CBT has been shown to be a highly effective short term psychotherapeutic intervention for a wide range of disorder groups, especially recent developments in cognitive behavioural therapies for treatment resistant schizophrenia and severe and enduring depressive disorder and their increasing positive evidence base initiated the prospect for the development of psychological interventions for bipolar disorders. To date there have been several efficacy studies and some experimental trials reporting on the effectiveness of adapted CBT in bipolar disorder. Overall these preliminary findings are promising and support the feasibility and clinical effectiveness of CBT for individuals suffering from bipolar disorder.

The relatively late development of psychological therapies for bipolar disorder might be due to the historical predominance of a biological paradigm in this disorder group, research investigating genetic and biological factors has been dominant, and there seemed to be a common misconception that most patients with bipolar disorder make a full inter-episode recovery. Secondly, earlier psychotherapeutic approaches for bipolar disorder came with the warning that patients suffering from bipolar disorder were poor candidates for psychotherapy, as they lacked sufficient insight, showed a high degree of dependency and formed poor therapeutic relationships.

II Bipolar disorders

2.1 Concept and phenomenology

One of the key features that led Emil Kraepelin to differentiate manic depression from schizophrenia at the beginning of the last century was the noted relatively more positive prognosis for individuals suffering from this recurrent mood disorder (Kraepelin, 1921, p.3). We now know that this presents a hugely simplified view. There is a need to differentiate conceptually key elements of both disorder groups and clinical presentations in mixed and comorbid states that influence onset and course of the disorder.

It is difficult to gain an accurate impression of the naturalistic course of bipolar disorder and the development of mood variations over the life-span. The observation of course and outcome in bipolar disorder generally includes the largely unquantified effects of routine and prophylactic treatments. Many studies of bipolar disorder focus on hospitalisations as key indicators for recurrence. This introduces a strong selection bias that results in a possible underestimate of recurrence as these studies will exclude individuals who recover without hospital admission. Studies focusing on the general population on the other hand often include a range of sub-syndromal presentations and subjective estimates of the severity and frequency of mood changes. Further, many historical population based studies of bipolar disorder included individuals suffering from recurrent unipolar depression.

Bipolar disorder is characterised by affective highs and lows, although states combining both manic and depressive symptoms are common. Symptoms are often recurrent throughout life with high rates of relapse (Gitlin *et al.*, 1995). Not surprisingly, bipolar disorder is associated with significant impairment in both social and occupational functioning, higher divorce rates, alcohol abuse, self harm and suicide (Goodwin and Jamison, 1990; Coryell *et al.*, 1993; ten Have *et al.*, 2002). Furthermore, the impact of bipolar disorder extends beyond the individual, often having a major impact on partners and families of sufferers (Dore and Romans, 2001). The disorder also has marked social costs in terms of treatment, social care and economic productivity, as many careers are often destroyed by the illness. Recently, Das Gupta and Guest (2002) estimated the annual burden of bipolar disorder to the NHS to be £199 million and the overall annual cost to UK society at £2 billion.

Although bipolar disorder is commonly described as a biological or genetic disorder (Craddock and Jones, 1999) there is increasing evidence that pharmacological treatments alone are often sub-optimal (Tondo *et al.*, 2001). This observation, combined with growing research which suggests that psychosocial factors play an important role in the onset and course of the disorder (Johnson and Roberts, 1995; Johnson, 2005), has led to the development of psychological models of bipolar disorder and combined medical and psychosocial treatment approaches.

2.2 Psychopathology and assessment

The criteria that help clinicians to differentiate unipolar from bipolar mood disorders have changed over time. In 1957 Leonhard proposed the essential feature of past mood

episodes to be included in the classification of mood disorders. Individuals with bipolar disorder or manic depression as it was coined then had to have a history of both mania and depression whereas individuals with unipolar depression only had to have a history of episodes of exclusively low moods. Bipolar patient groups however are also heterogeneous and include different courses, pattern or recurrence and cycles. Goodwin and colleagues suggested in 1976 that bipolar disorder should be classified as bipolar I and bipolar II disorder (Dunner *et al.*, 1976). Bipolar I were defined as those patients with a history of mania, usually severe enough to merit psychiatric treatment and often accompanied by features of psychosis, and bipolar II patients had in addition to a history of depression episodes of hypomania, severe enough to be noticed as abnormal by the patient or their close others resulting in an interference in normal functioning but not as severe as requiring hospital treatment or long term psychopharmacological treatments. One can easily see how the line between these two sub categories remains vague.

In sum, any description of bipolar disorder contains periods of clearly described and observable affective highs and lows which can often co-occur simultaneously in mixed states (Goodwin and Jamison, 2007). Symptoms of mania usually include a subjective heightening of mood or excitement, irritability and even anger, increased physical activity, more and faster speech, feelings of increased energy with little need for sleep, increased mental activity, racing thoughts and feelings of heightened perception. Symptoms can range from mild hypomania through to more severe manic episodes where patients report hallucinations, delusions and other psychotic phenomena (Carlson and Goodwin, 1973).

As in unipolar depression bipolar depressive states are defined by a decrease in subjective mood and a slowing of behaviour and cognition. Feelings of worthlessness, guilt and

suicidality are common; other symptoms include loss of interest in activities, disturbed sleep, agitation and poor concentration. These may range from mild affective changes with few apparent symptoms and little impact on daily life to profound depressive episodes marked by hallucinations, extreme psychomotor retardation and severely impaired cognition, any of which may require hospitalisation (Goodwin and Jamison 2007).

In most clinical descriptions mania and depression in bipolar disorder are described as mutually exclusive. These descriptions underestimate the frequency of co-occurrence of manic and depressive mood states in so called mixed episodes. Cassidy and colleagues described in 1998 in a factor analytic study of 237 manic and mixed (manic and depressed) patients that the strongest factor suggested that dysphoria, rather than euphoria, was most commonly present in mania.

2.3 Diagnosis and definitions of bipolar disorder

Current classification of bipolar disorder is defined by the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR: American Psychiatric Association, 2000) and the World Health Organisation's International Classification of Diseases version 10 (ICD-10; World Health Organisation, 1992).

Longitudinal research in the course of bipolar disorder provided support for spectrum of severity within bipolar disorders ranging from subsyndromal Cyclothymia, to Bipolar II

disorder through to Bipolar I disorder (for an overview see: Goodwin and Jamison, 2007). The different classifications are described as follows:

Table 2.1: Classification of bipolar affective disorder

Bipolar I	evidence of at least one manic or mixed episode in a patient's psychiatric history. A major depressive episode is not required for this diagnosis.
Bipolar II	requires that an individual has had at least one hypomanic episode and at least one major depressive episode within their psychiatric history. They must not, at any time, have met criteria for a manic or mixed episode.
Rapid cycling	four or more mood episodes occur within any 12 month period.
Cyclothymic disorder	an individual must have experienced "numerous periods" of hypomania along with periods of depressed mood or loss of interest, none of which meet criteria for major depressive episode within the last 2 years (1 year in children or adolescents). During that period, the individual must not have been symptom-free for longer than two months. The person must never have had a manic or mixed episode and symptoms must cause clinically significant distress or impairment in functioning.
Bipolar disorder NOS	disorders with bipolar features that do not meet criteria for any other bipolar disorder. These can include a variety of presentations such as rapid alternation between manic and depressive symptoms, which meet symptom threshold but not duration criteria, recurrent hypomania with no depressive history or a manic or mixed episode superimposed on another major psychiatric condition.

In line with the recent reconceptualisation of mental health disorders it has been suggested that bipolar disorder is best described as a spectrum of disorders, sharing phenomenology and aetiology (e.g. Angst *et.al.*, 2003). Any bipolar spectrum description ought to include a description of hypomanic personality (Kwapil *et.al.*, 2000) and of the bipolar subtypes from cyclothymia through to bipolar I disorder, including descriptions of common mood states, depression, hypomania, mania and any observable combination.

Hence there is an ongoing debate as to whether bipolar disorders should be viewed as discrete disorders or as a disorder which varies by degree (Goodwin and Jamison, 2007). In the current research literature however dimensional models of bipolar disorders are prevalent in an attempt to distinguish key clinical features. More recently high-risk methodologies have received increased attention in the study of bipolar disorders (e.g. Depue *et al.*, 1989; Meyer and Hautzinger, 2003). This method essentially involves identifying a population who have not developed the disorder but possess pathological indicators which suggest they are at increased risk of doing so. Pathological indicators may be genetic (i.e. genetic markers in family members of a bipolar patient), cognitive (such as the attributional style in the theory of hopelessness depression), or behavioural symptoms such as those outlined in Depue (1981) or Eckblad and Chapman (1986). This approach has provided important theoretical and clinical understandings of more severe forms of the disorder (e.g. Depue *et al.*, 1981; Eckblad and Chapman, 1986; Meyer and Hautzinger, 2003).

Johnson and colleagues recently put forward that bipolar disorders could helpfully be reconceptualised in a pre Kraepelinian way and defined as unipolar mania with co-occurring major depression as a common but not necessary feature (Johnson, 2005). This notion is based on the argument that unipolar and bipolar depression are essentially

indistinguishable (Cuellar *et al.*, 2005) and that mania often occurs without an associated episode of depression. Further, that in psychological terms mania and depression in bipolar disorder do not share the same mechanisms in that the triggers for mania (sleep/circadian/social rhythm disruption or goal attainment life events), and depression (negative events, cognitive style, self-esteem), (e.g. Johnson and Meyer, 2004; Cuellar *et al.*, 2005) are essentially different and that they are not necessarily of the same aetiology.

2.4 Onset and course of the disorder

The disorder course is commonly described as chronic, and although there is considerable inter individual variation it is typically one of acute episodes of mania and depression interspersed with periods of relative stability. A striking finding from many studies is that even between mood episodes, individuals experience significant subsyndromal symptoms (Judd *et al.*, 2002; Judd *et al.*, 2003). In a 13-year longitudinal study of Bipolar I patients, Judd *et al.* (2003) found that patients, supposedly in remission, experienced subsyndromal depressive or hypomanic symptoms nearly 40% of the time. Moreover, bipolar disorders are often associated with other co-morbid disorders. In a large population study of Bipolar I patients, Kessler and colleagues (Kessler *et al.*, 1997) found that many patients reported at least one other DSM-III disorder and nearly 60% stated that this was present before the onset of bipolar disorder. This suggests that individuals with bipolar disorder suffer from complex emotional difficulties.

The onset of more severe forms of the disorder tends to occur between 15 - 24 years of age, with peak age of onset between 15-19 years (Goodwin and Jamison, 2007). However, many researchers have suggested that milder forms, such as cyclothymia,

appear earlier in adolescence (Akiskal et al., 1977; Depue et al., 1981) which is associated with an increased risk of developing more severe forms at a later stage.

Relapse and recurrence in bipolar disorder is frequent. Even with state of the art psychopharmacological maintenance treatments the risk of relapse into mania or depression within 2 years is around 38% (Gelenberg et al., 1989) and relapse within 5 years stands at around 73% (Gitlin et al., 1995). Although other prospective studies suggest that relapse is no more frequent than in unipolar depression, relapse in bipolar disorder is more often associated with work impairment, lower functioning and hospitalisation (Goldberg et al., 1995).

2.5 Psychosocial factors

A growing body of evidence suggests that the current environmental context has an important impact on the onset, course and expression of bipolar spectrum disorders (Alloy et al., in press a; Alloy, Reilly-Harrington, Fresco, & Flannery-Schroeder, in press c; Johnson & Roberts, 1995; Johnson & Kizer, 2002). The role of two kinds of environmental factors has been studied in bipolar disorder: recent life events and social support (including negative support such as expressed emotion). The life events literature has been fairly consistent in suggesting that bipolar individuals experience increased stressful events prior to onset or subsequent episodes of their disorder. In addition, there is reasonable evidence that social support from significant others leads to a more positive course of bipolar disorder, whereas negative support (e.g., high expressed emotion) from family and friends predicts a worse course of bipolar disorder.

Psychosocial factors are those factors, by definition, which have a psychological and a social basis. They can be conceptualised as social factors that have an influence on psychological wellbeing and in turn psychological factors that impact upon social functioning. This is very simplified and it is likely that the interplay between factors is complex and affected by numerous external factors at one time. Though the factors will be discussed more or less in isolation they can be considered as moderators and mediators of outcome in bipolar disorder.

Psychosocial impairments are thought to be apparent for a number of years following a severe episode requiring hospitalisation (Coryell et al., 1993; Goldberg & Harrow, 2004), with 30-60% of individuals never regaining full social and occupational functioning (MacQueen et al., 2001). Fagiolini et al. (2005) found that the degree of functional impairment sustained during remission in bipolar disorder patients was correlated with the degree of depressive spectrum symptoms. They went on to suggest that patients who report depressive subsyndromal symptoms may benefit from a comprehensive psychosocial and rehabilitative intervention. They indicated that not only could this improve patients' level of disability, it also has the potential to improve their quality of life and reduce the number of future episodes. In a large longitudinal study, it was found that in Bipolar I individuals, as depressive symptom severity increases, there is a significant increase in psychosocial disability which does not decrease significantly in asymptomatic periods. However, patients with Bipolar II actually experience a slight improvement in psychosocial functioning when they are hypomanic (Judd et al., 2005). Impairments compared to a non-bipolar population are widespread. Psychosocial impairments are rife as regards income, occupational status, and education. Individuals with bipolar illness are half as likely to be married and if married are twice as likely to be divorced or separated (Coryell et al., 1993).

It has been suggested that the association found between poor outcome and low social class, inadequate social support and unmarried status are a result of a lack of psychological and economic buffering normally utilised in stressful situations, i.e. that there are common vulnerabilities that lead to both illness and psychosocial dysfunction. However, it is equally plausible that these factors are simply illustrations of the impact bipolar episodes have upon individuals (O'Connell et al., 1991).

Various factors that have been implicated in poor functional outcome include pre-morbid functioning (MacQueen et al., 2001); cognitive impairment (Martinez-Aran et al., 2004, Zubietta et al., 2001); age at first episode (Perlis et al., 2004); number of previous affective episodes (Tohen, Waternaux & Tsuang, 1990) though this has not always been found (Yan et al., 2004); number of previous hospitalisations (O'Connell et al., 1991); and comorbidity with other Axis I disorders.

The direction of causality is difficult to ascertain between psychosocial and psychological factors and bipolar disorder. Indeed it is likely it is a bidirectional, fluid relationship. The majority of research has been carried out looking at individuals who have already experienced bipolar episodes, where impairments in these factors could be a cause or a consequence, or a combination of both. Prospective longitudinal studies of individuals at risk of developing bipolar disorder may allow us to understand this complex relationship.

2.5.1 Life events and difficulties

Major life events, e.g. giving birth, death, family disruptions, redundancy etc, have been suggested as precipitating affective episodes (Kessing et al., 2004; Christensen et al.,

1993). Recent life events have been found to be associated with suicide in individuals with bipolar disorder (Isometsä et al., 1995). Most therapies developed for bipolar disorder, particularly CBT, family therapy and IPT/IPSRT, include aspects to help develop skills to cope with stressful events more effectively. Life events studies have been limited by methodological problems such as poor questionnaire design, and a lack of control group, often comparing time periods before episode onset and with control periods.

Research has examined the experience of stress for different diagnostic groups. Perris (1984) in a retrospective study, found that people with bipolar disorder experienced more 'independent' life events (unrelated to depression) than those with unipolar disorder, but less than those with neurotic disorders. Myin-Germeys et al. (2003) who sampled stressors, cognitions and mood at random times, found more activity-related stress in bipolar patients in remission following minor subjective stressful life events, compared to major depressed and control groups. Examining a younger sample, Tillman et al. (2003) found that youths with bipolar disorder experienced more stressful life events than youths with attention-deficit hyperactivity disorder (ADHD) and normal controls. In a nonclinical sample, cyclothymic individuals experienced more daily stressors than those with intermittent depression or healthy controls (Lovejoy & Steuerwald, 1997). However Swann et al. (1990) found that there were no significant differences in the perceived role of stressful events as a function of diagnosis (unipolar or bipolar). Chung et al. (1986) conducted a retrospective study comparing hypomanic patients with patients with schizophrenia and schizophreniform psychosis. In the 26 weeks before onset, 14% of hypomanic patients experienced a 'threatening' event compared with 33% of the schizophrenia group and 66% of those with schizophreniform psychosis. The differences between the rates of life events in the schizophreniform group and hypomanic group were significant, although there were some demographic differences between the

two groups in terms of age and employment status. Bebbington et al. (1983) also compared diagnostic groups in 97 patients in the Camberwell Collaborative Psychosis Study who were admitted to hospital. They found that there were significant differences in the number of life events experienced in the months before onset, with individuals with psychotic depression experiencing the most events, followed by manic, schizophrenic, and the least experienced by controls. Thus, although most studies appear to point to a role played by life events, may not be as important as in other psychiatric conditions.

A number of studies have pointed to an effect of life events on the course of the disorder. In recent thinking, the influence of life events has been attributed principally to early episodes (McPherson et al., 1993). Later in the course of illness there is mixed evidence of the importance of life events in risk of relapse. Ambelas (1987) found that those experiencing their first manic episode were significantly more likely to have experienced life events in the 4 weeks prior to admission than those patients in repeat episodes. However assessment of life events was judged retrospectively in this study, a common methodological problem of many studies examining life events. Other early studies found that up to 50% of patients recalled a major life event preceding their initial episode of bipolar disorder (Dunner et al., 1979; Glassner & Haldipur, 1983).

A number of prospective studies found increases in life events in the weeks or months prior to relapse (Hunt et al., 1992; Christensen et al., 2003; Hammen & Gitlin, 1997; Cohen et al., 2004), whilst others found no such association despite having similar and stringent methods (McPherson et al., 1993). It is perhaps noteworthy that the sample in the McPherson et al. study from Dunedin in New Zealand was more affluent than Hunt et al's (1992) patients from inner-city London and was at a later stage of illness.

Bidzinska's (1984) retrospective study of 97 patients with affective disorder found that later phases of bipolar disorder have significantly fewer life events (as assessed by review of medical charts) than earlier episodes. There were no significant differences for unipolar illness. Conclusions drawn from these findings are that the significance of life events in the onset of a bipolar episode is much greater in the earlier stages of the illness, and that in later stages the impact of life events is perhaps mediated by other factors including number of previous episodes, age, or socio-economic status.

The kindling/behavioural sensitization model developed by Post (1992) seeks to explain the reduced predictive impact of stressful life events over time. Post's theory suggests that individuals with affective episodes become more independent of life stressors as number of episodes increase. The model can be separated into two distinct, although conceptually-linked, theories; kindling; and behavioural sensitisation. The kindling component of the model hypothesises that the illness becomes autonomous after a certain number of episodes and stressors. The sensitisation aspect of the model is intrinsically different, theorising that the patient has increased sensitivity to life stress as the illness progresses, to the point where very small amounts of stress can precipitate a relapse. This perhaps ties in with Goodwin & Jamison's (1990) finding that the time between episodes shortens as duration of illness lengthens. Both theories point towards increased chronicity as the course of illness progresses, with patients becoming more prone to relapse, independent of life events, over time. The discrepancy between the two parts of Post's model permits different interpretations of results, such that one set of findings could be construed to support or refute the model (Hlastala et al., 2000).

Thus it comes as no surprise that evidence for Post's hypotheses is mixed. In Hammen & Gitlin's (1997) longitudinal study, patients with a higher number of episodes were more

likely to relapse, and were found to relapse more quickly, following a stressful life event than patients with fewer episodes. The authors concluded that their findings disconfirm Post's kindling hypothesis as the patients with more episodes did not have "autonomous" episodes. An alternative conclusion that could be drawn from the same findings is that the threshold for an event to be stressful has been lowered in patients with more episodes thus they were more likely to relapse after an event, and also relapse quicker, therefore supporting Post's sensitisation hypothesis. The model remains contentious and continues to be interpreted in a variety of ways by researchers (Hlastala et al., 2000).

Swann et al. (1990) found that people with high environmental stress had a longer index episode and fewer previous episodes than those who reported low stress, thus supporting the kindling hypothesis. This group found no effect of age. An alternative hypothesis, as suggested by Swann and colleagues, is that there exists a stress-sensitive subgroup of depressed patients who have fewer episodes than a more autonomous group. However, results were for a sample of individuals with affective disorder, and not specifically bipolar.

In a study of 64 bipolar I patients, Hlastala et al. (2000) examined life events in the three months before an index episode and during a three month episode-free period. The number of episodes experienced was not able to predict the level of stress experienced before an episode. This finding does not support Post's kindling hypothesis that later episodes are expected to be associated with a relative absence of events. Post's sensitisation hypothesis was not supported either as lower levels of stress were not observed in the later episodes. A complex relationship was observed between age and levels of stress. As age increased, there was greater likelihood of experiencing a low level of stress, and a lower probability of experiencing a high level of stress prior to episode

onset, independent of number of episodes. These findings suggest there is perhaps something specific about the aging process that mediates the relationship between stress and episode onset e.g. social support or coping strategies.

Kim et al. (2007) also found an effect of age on the relationship between life stress levels and mood symptoms, albeit in a sample where all patients were relatively young. The researchers investigated the impact of life stress on the course of early-onset bipolar disorder in a sample of adolescents. They found that chronic stress in relationships, particularly romantic relationships, was associated with less improvement in all types of mood symptoms. They also found higher levels of chronic stress, and higher severity of independent events, were more strongly associated with mood symptoms among the older adolescents in the sample. Further analyses revealed that age was not correlated with the frequency or severity of independent events, suggesting that older age is not simply associated with greater severity of life events. However it is not clear whether the older adolescents had longer illness histories or more previous episodes, so we can not conclude whether the effects are due to age or stage of illness. Nevertheless, these findings are consistent with Post's stress-sensitisation hypothesis, predicting an increased sensitivity to life stress as the illness progresses. However whether the increased sensitisation occurs as a result of actual illness progression or age may still be up for debate.

Having established that there is some evidence to suggest that life events precede at least early episodes of bipolar disorder, we turn our attention to the effect stressful life events have on outcome. Ellicott et al. (1990) in a longitudinal study examined the impact of life stress on 61 bipolar outpatients over two years. Survival analysis showed an association between life events and relapse, with medication and compliance controlled for. There

was, however considerable variability in relapse and in the experience of stress. This study was therefore followed up by Swendsen and colleagues (1995) in another longitudinal study which compared the clinical and psychological characteristics of stress “reactive” individuals with those who are more “resilient”. High stress, which was associated with the personality characteristics of extroversion and obsessionality, was found to be a predictor of relapse. Johnson & Miller (1997) examined the effects of life events on recovery using monthly interviews. They found that individuals experiencing a severe life event during the index episode took three times as long to recover.

In addition there may be a differential effect of life events on type of relapse. Christensen and colleagues (2003) found an increase in reported life events using questionnaire assessment prior to depressive episodes but not manic episodes. Gender differences were found, with a significant number of depressive episodes in women, but not men, being preceded by negative life events. However, as the authors state, this may be because women have a higher awareness of psychosocial stressors. In contrast, in a prospective study, Pardoen et al. (1996) found no association between recent life events and the onset of depression. The authors did find that bipolar patients with a manic/hypomanic relapse had more marital stressors prior to relapse than other bipolar patients. However, this study had a small sample of bipolar patients ($n=27$). Hunt et al. (1992) completed a longitudinal 2-year study, using interview assessment which helps to reduce mood related bias. No difference was found in rate of life events for depressive and manic relapses.

Since the 1970s researchers have begun to specifically examine the relationship between life events and mania. In a matched-controlled study assessing life events prior to admission, Kennedy et al. (1983) found that a sample of 20 manic patients had

experienced three times as many independent events rated objectively as having a negative impact than controls. Assessment of life events was retrospective. In a study with a matched control design, 14 manic patients retrospectively experienced more uncontrollable and unanticipated life events than 14 bipolar patients who had not experienced a mania, but there was no significant difference in the number of events. Sclare & Creed (1990) in another retrospective study, found no relationship between the onset of mania and life events. This study, as with many of the early studies, suffered from a small sample size ($n=30$) and in addition there was no control group. Babington et al (1993) found an excess of life events in the months prior to onset of mania. Life events were not limited to severe, but also included mild life events. The increase in life events rated as having a moderate to large degree of threat was significant compared to controls. Both the prior studies used the Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978) which is seen as a sophisticated instrument in the assessment of life events whereas other studies used modifications of the LEDS (Kennedy et al, 1983; Ambelas, 1987). However, again samples were small. In a large scale Danish study of 1565 patients and 31,300 controls Kessing et al (2004) found a significant risk of first admission with a diagnosis of mania following a major life event such as suicide of mother or sibling and recent divorce or marriage. Chung et al (1986) found a link between life events blindly rated as threatening, and the onset of hypomania. The number of threatening life events experienced by hypomanic individuals, although double that of the controls, did not reach statistical significance.

Life events involving goal attainment have been found to be particularly significant in the onset of manic symptoms. The Behavioural Approach System (BAS) dysregulation theory (Depue, Krauss & Spoont, 1987) is a prominent biopsychosocial theory of bipolar disorder which aims to explain the mechanism by which this happens. According to this

theory, bipolar patients are thought to demonstrate an excessive increase in BAS activity in response to BAS activation-relevant events e.g. goal striving, which is reflected in hypomanic and manic symptoms. In other words the reward involved in goal attainment events stimulates the oversensitive behavioural activation system which enhances positive affect to facilitate goal-directed behaviour. Conversely, an excessive decrease in BAS activity is thought to occur in response to BAS deactivation-relevant events e.g. failure. Thus depression is the result of an inactive BAS failing to produce positive affect or incentive-reward motivation. It is this dysregulation, or variability, in the system that is theorised to make bipolar individuals more sensitive to environmental experiences. The model suggests that individuals who exhibit the greatest baseline variability in mood will be the most vulnerable to the effects of life events (Johnson & Roberts, 1995).

The BAS dysregulation theory makes specific predictions about the types of life events that trigger the onset of bipolar episodes and symptoms, suggesting that depression and mania are precipitated by different types of environmental events (Johnson & Roberts, 1995). This theory does not receive support from some studies above (e.g. Swann et al., 1990; Hunt et al., 1992) which showed that negative events could trigger both depression and mania. However many other studies tend to cluster stressful life events together, rarely examining the impact of different types of life event.

This nascent theory has seen a very recent upsurge in studies looking into life events and the onset of mood symptoms from the BAS perspective, with mixed results. Using a life events interview, Johnson et al. (2000) found that manic but not depressive symptoms were increased in bipolar I individuals in the 2 months following goal-attainment events. This increase in symptoms did not occur for general positive events. Nusslock et al. (2007) also found, consistent with the BAS dysregulation theory, that a goal-striving event

(preparing for and completing final exams) was associated with an increase in hypomanic but not depressive episodes and symptoms in individuals with a soft spectrum diagnosis. This relationship was not reduced when sleep loss was controlled for. The sample in this study was drawn from college students with bipolar II disorder or cyclothymia suggesting BAS dysregulation can be observed in the spectrum of bipolar disorder, not just in bipolar I disorder. However caution should be taken in generalising these findings to the whole spectrum. It is plausible that goal-striving events are more salient to individuals in further education, such as in this sample. This group may also have particular protective factors that differentiate them from other bipolar patients in the community e.g. high intellect, high socioeconomic status.

Meyer et al. (2001) examined whether self-reported BAS sensitivity alone, rather than the perception of incentive (e.g. goal-related events) would place bipolar patients at more risk of mania. They found significant relationships between self-reported BAS scores and manic symptom intensification over time, suggesting that BAS sensitivity may represent a vulnerability to mania in bipolar disorder. BIS self-reports were correlated with depressive symptoms, although did not predict increased depression over time. This suggests that BIS self-report levels are a state-dependent characteristic of depression. Salavert et al. (2007) also examined the functioning of both BAS and BIS systems, and their influence in subsequent episodes. On study entry, 39 euthymic bipolar I patients showed higher BAS scores than the group of 38 controls suggesting a somewhat trait-related vulnerability. Eighteen months later, patients relapsing with a depressive episode had lower BAS scores than patients with a manic/hypomanic episode, and a tendency to score lower than patients still euthymic. However unlike the Meyer et al. (2001) study, BIS scores did not differ.

These studies seem to support, to varying degrees, the BAS dysregulation theory. However Biuckians et al. (2007) found, contrary to hypotheses, adolescents with high BAS levels exhibited less severe concurrent mania symptoms. Furthermore levels of BAS sensitivity were not associated with concurrent levels of depression. These findings suggest that BAS functioning is inversely related to mania symptomatology in bipolar adolescents. However the sample size was small ($n=25$) and the study cross-sectional in design. It would have been interesting to examine whether the BAS/BIS levels were predictive of future episodes, and whether they changed over time.

A number of other theories aim to explain the mechanism by which life events affect mood. Life events have been shown to be correlated with response to medication. Kulhara et al (1999) found that lithium non-responders had significantly more life events and total stress, as well as less social support. The authors hypothesise that increased life events and stress, in turn decrease social support, decrease lithium compliance and therefore increase likelihood of relapse. A limitation of this study was the cross-sectional assessment of psychosocial factors and retrospective assessment of life events. However, Johnson & Miller's (1997) finding that major life events have been found to take 3 times as long to recover from an affective episode was not mediated by medication compliance. Ellicott's (1990) study also found that the relationship between life events and relapse remained, when medication and compliance were controlled for. This suggests that the psychosocial impact goes above and beyond the biological and that its mechanism of action is not through disruption of medication compliance (Johnson & Miller, 1997).

The 'stress-diathesis model' integrates experience of life events with a biological vulnerability to explain onset of affective episodes (Goodwin & Jamison, 1990). This theory proposes that individuals with bipolar disorder have an inherent biological

vulnerability, as a function of circadian rhythms. Circadian rhythms are regular changes in our mental and physical characteristics throughout the day. They can be affected by external factors and at an extreme disrupted. Such disruptions have been postulated as a risk factor for affective episodes (Goodwin & Jamison, 1990; Wehr et al., 1987). For instance sleep deprivation resulting from a life event consequently increases the risk of relapse into a manic state. Kadri et al. (2001) found that relapses were more likely to occur in (Muslim?) bipolar patients in the month of Ramadan which involved a disruption to eating habits. Columbo et al. (1999) found that around 10% of bipolar depressed individuals subjected to sleep deprivation switched into mania or hypomania. Mania itself maintains the disruption of circadian processes by increasing the likelihood of further insomnia, thus becoming a vicious circle (Wehr et al., 1987). In turn the consequences of the disorder may themselves produce life events, which in turn will continue to produce further events (Christensen et al., 2003).

A related stress-vulnerability model is the social zeitgeber theory of mood disorders (Ehlers et al., 1988). According to this theory, life events produce changes in social zeitgebers (persons, social demands or tasks that set the social clock) which, in turn, lead to social rhythm irregularity. This instability if coupled with other vulnerabilities then disturbs biological rhythms and leads to a mood episode. Even life events which are apparently psychologically non-stressful can place considerable stress on the body's attempt to maintain synchronised biological rhythms (Frank et al., 2006). In support of this theory, Ashman et al. (1999) found that patients with rapid cycling bipolar disorder had less rhythmic daily routines and completed fewer activities on a regular basis than control subjects.

The original social zeitgeber theory as proposed by Ehlers et al. (1988) was based on the association between life events and depressive symptoms. As examined above, life events are also associated with manic and hypomanic symptoms, and subsequent studies have sought to corroborate this relationship with the theory. Malkoff-Schwartz et al. (1998) found that the number of social rhythm disrupting events was different preceding depressive and manic episodes. There were substantially more manic individuals who had experienced at least one event prior to onset than depressed patients. It was suggested this may be due to the more gradual social rhythm disruption involved in depressive episodes, which therefore requires a longer period of study than the 8 week pre-onset period in this study. The same authors conducted a similar study to examine this further, but with a longer pre-onset period to each participant's mood episode onset (Malkoff-Schwartz et al., 2000). Again manic patients were found to have experienced more disruptive events in the pre-onset periods than their control periods compared to bipolar depressed, bipolar rapid cycling, and unipolar depressed groups. Once more only the manic group was found to experience significantly more disruptive events in the 8 weeks prior to onset compared to the 8-week control period. The null findings for the 20-week pre-onset period dispute the notion of a delayed effect of disruptive life events on depressive episodes. It would appear that the success of treatment strategies aimed specifically at helping bipolar patients maintain regular routines in the face of stressful life events (e.g. Frank et al., 2005) support the social zeitgeber theory.

It is likely that the effect of life events is moderated by other psychosocial factors, such as cognitive factors and social support. Lyon, Startup & Bentall (1999) found that bipolar-depressed patients attribute negative events to themselves and make more global and stable attributions for negative events. They found that while manic patients made self-serving attributions to fictional events, they also attributed negative events to self.

However, another study found no difference to the attributions made about life events between bipolar and controls (Tracy et al, 1992). Another suggestion is that stressors have a differential effect on outcome dependent on type of cognitive vulnerability. In an 18 month follow up of remitted bipolar patients, Hammen et al. (1992) found that symptom severity was significantly associated with sociotropy, interpersonal events and an interaction of the two. There was no similar effect for autonomy or autonomy/achievement events. Kennedy et al. (1983) found that manic patients were less likely to have an adequate confiding relationship prior to admission. This subset of patients had significantly more undesirable events.

Heredity has also been suggested as a biological mediator between life events and relapse. Johnson et al. (2000) found that life events in bipolar patients had a differential effect depending on family history of affective disorder. Bipolar patients with heredity had a lower age of onset, and less life events prior to onset. The prevalence of life events decreased as number of episodes increased, which provides support for the kindling hypothesis. More research is required into the question of whether there is an underlying vulnerability which causes emotional-stress sensitivity, or whether the disorder or mood itself causes a change in behaviour or reactivity. Longitudinal designs of major life events are required to examine the mechanisms further.

Further research should address methodological problems such as retrospective designs and unreliable methods of life event assessment, prevalent in the older studies. Johnson (2005) argues that a design which compares life stress prior to and after episodes, is more valid than comparing bipolar individuals, who invariably have a high level of life stress even in asymptomatic periods, with individuals with no mental illness. She also asserts there is a need for a distinction between the life-events and episode link, and the

link between life-events and increasing symptomatology. Additionally, many studies do not differentiate between life events that are independent, or dependent on people's behaviour, and therefore does not differentiate between risk factors and causes of bipolar disorder (Alloy et al., 2005). Sclare & Creed (1990) argue, that many studies finding an elevated number of life events prior to onset of episodes, are picking up on prodromes of mania or depression. As raised by the authors, this becomes significant when the beginning of onset is taken as the date of hospitalisation, which is often much later than the beginning of symptoms. Furthermore, with few exceptions, studies do not differentiate between different types of life events e.g. positive and negative. There may be merit in examining if different types of life event precede different types of episode e.g. it is suggested that marital events are more likely to precipitate manic relapse, and it could be theorised that negative life events such as bereavement precipitate depressive relapse. In a recent psychosis study which separated the different types of stressful life events Kim et al (2007) study showed family, romantic and peer relationship stress showed strongest association with mood symptoms.

Research could also usefully address the question of which individuals are more vulnerable to stressful life events. Aronson & Shukla (1986) found that patients who relapsed after a hurricane had experienced a shorter period of stability prior to this, in terms of subsyndromal symptoms, interpersonal stressors or concurrent personality disorders. Identifying individuals at risk of further episodes will inform clinical work, and indicate where research should be targeted.

In summary, stressful life events have been shown to affect both the onset and to a lesser extent the course of bipolar disorder. However, the specific nature of this relationship, and the mechanisms behind it, have yet to be fully understood.

2.5.2 Childhood Stressful Life Events

Having reviewed the literature implicating stressful life events in the onset and recurrence of bipolar disorder episodes, we turn to examine the role that childhood life events play in the development of bipolar disorder and the impact they have on its course.

Until recent times, few studies had investigated the potential role of early life stresses on the subsequent course of bipolar illness. Bauer et al. (1997) followed 103 bipolar patients enrolled in a 1-year treatment program with the aim to identify predictors of service utilisation. The sample included patients with comorbid diagnoses and the only exclusion criterion was pre-existing moderate to severe dementia. A recalled history of childhood physical abuse was found to be one of only two predictors of service utilisation, the other being the presence of a major affective episode at clinic intake.

In a cross-sectional study, Levitan et al. (1998) observed a strong relationship between mania and childhood physical abuse, with bipolar individuals having a significantly greater rate of childhood abuse than depressed patients. However, as is problematic with most studies in this area, both these studies used retrospective reporting for the disclosure of information regarding abuse.

Leverich et al. (2002) explored the association of abuse in childhood or adolescence with course of bipolar disorder and illness characteristics. In this study 631 outpatients with bipolar disorder were evaluated using both cross-sectional and longitudinal study design. Altogether 49% of women and 36% of men in the sample reported early abuse in childhood or adolescence, exceeding statistics reported in the general population

(Finkelhor et al., 1990; MacMillan et al., 1997). History of physical or sexual abuse was predictive of a more severe course of bipolar illness. In particular, patients who reported abuse had earlier onset of illness, an increased number of comorbid disorders, faster cycling frequencies, a higher rate of suicide attempts, and more psychosocial stressors occurring before the first and most recent episodes. Patients who reported a history of physical abuse showed a significant pattern of increasing severity of mania, supporting the relationship between childhood physical abuse and mania reported by Levitan et al. (1998). Although the results are promising in terms of elucidating a relationship between early traumatic experience and course of bipolar illness, the authors warn of making assumptions of a causal relationship. A possible interpretation of the data is that early behaviours associated with a poor course of illness could promote harsh discipline and provoke the early abuse (Friedrich & Boriskin, 1976).

Goldberg & Garino (2005) examined the extent of posttraumatic stress disorder (PTSD) in relation to past abuse in bipolar adults, and its impact on the course of illness. They found that 51% of their sample of bipolar adults reported severe childhood abuse, and of those around 35% had PTSD. As expected, this was significantly more than the group who denied any childhood abuse. Furthermore, risk of developing PTSD had a linear relationship with the number of subtypes of childhood abuse or neglect present (e.g. emotional, physical, sexual). No statistical differences in the course of the bipolar illness were found between the PTSD and non-PTSD groups, although variables such as cycle frequency and severity were not measured. The authors acknowledge that further studies are needed to elucidate the role of childhood trauma in the development of bipolar disorder independent from PTSD.

Garno et al. (2005) also found that childhood abuse was reported in about half of their sample of 100 bipolar adults, with multiple forms of abuse reported in around a third. Consistent with the Leverich et al. (2002) study, negative outcome was significantly associated with history of abuse. Specifically, younger age at onset, increased lifetime suicide attempts, rapid cycling, and comorbid substance misuse were significantly associated with various and multiple forms of abuse. Interestingly, the study found differences in illness outcome across abuse subtypes. In particular, a significant association was found between lifetime suicide attempts and severe childhood sexual, but not emotional or physical, abuse. Coupled with findings of previous studies which found a relationship between mania and childhood physical abuse (Levitan et al., 1998; Leverich et al., 2002) there is suggestion of a polarity effect of subtype of abuse. However this requires further examination as other studies report a connection between childhood physical abuse and suicidal behaviour among wider psychiatric conditions (Silverman et al., 1996). In addition, Garno et al. (2005) also found that multiple forms of childhood abuse appeared to heighten the risk of suicide attempts.

In a sample of adolescents with bipolar disorder and controls, Rucklidge (2006) found that over 50% of the bipolar patients reported a history of trauma, consistent with the adult studies. This finding indicates that individuals with a history of trauma are likely to have experienced onset of their illness by adolescence, otherwise one would expect the figure to be lower in this adolescent group. This supports the findings of other studies which show that patients who experience childhood adversity have a significantly younger age of onset of illness (Leverich et al., 2002; Garno et al., 2005; Dienes et al., 2006). As regards psychosocial functioning, adolescents with paediatric bipolar disorder reported lower self-esteem, more hopelessness, more traumatic experiences and negative life events, greater difficulties in regulating emotion, and poorer coping strategies than

adolescent controls. However a history of trauma did not differentiate those with and without psychosocial problems.

Potential explanations for the association of childhood stressful life events and bipolar disorder were examined by Grandin et al. (2007). In particular, they researched two possible models: harsh environment; and stress generation effects. According to the harsh environment hypothesis, bipolar (and unipolar) patients report more negative life events because they actually experience more negative life events that play a part in the onset of their disorder (Safford et al., 2007). On the other hand, the stress generation effect hypothesises that patients generate more negative life events as a result of the characteristics and behaviours associated with their symptoms (Hammen et al., 1991). This is conceptually similar to Friedrich & Boriskin's theory (1976) that early behaviours associated with a poor course of illness could promote harsh discipline and provoke early abuse. It is also linked to the suggestion in Geller & Luby's (1997) review that childhood onset bipolar disorder is characterised by hypersexuality, which may in turn influence the incidence of childhood sexual abuse (Dienes et al., 2006). Naturally this argument is highly contentious. In order to assess the two opposing theories, which represent opposite causal directions, Grandin et al. (2007) discriminated between independent and dependent childhood stressful life events, and examined the relationship of each to bipolar diagnosis. They found that the total number of reported childhood events, particularly independent events, occurring prior to onset was associated with bipolar diagnosis, thus supporting the harsh environment hypothesis. Bipolar diagnosis was not able to predict total number of childhood stressors, particularly dependent events, occurring after the age of onset. The stress generation hypothesis was therefore not corroborated.

Following on from Post's (1992) theory of stress-sensitisation, another form of stress sensitisation proposed is early adversity sensitisation. Post et al. (2001) propose that early adverse events can permanently alter the stress response system, sensitising individuals to later stress, and contributing to early onset and severe course of illness. The theory of stress sensitisation hypothesises that patients with a greater number of episodes will relapse following mild and severe stressful events. The early adversity sensitisation hypothesis theorises that bipolar patients who have experienced severe childhood adversity will relapse under mild levels of stress, regardless of the stage of their illness. Dienes et al. (2006) tested this hypothesis with a sample of 58 adults with bipolar disorder. They found that, in support of the early adversity hypothesis, patients with a history of relatively severe childhood experiences had a higher likelihood of relapse following mild stress than patients with mild or no childhood adversity. However no support was found for the original stress sensitisation hypothesis as number of episodes failed to moderate the association between stress and relapse. The between-group design of the study may be partly to blame for not picking up on changes in the association. They also observed that patients who experienced early adversity had a significantly younger age of onset of illness, thus giving support to the theory that they are more likely to have their first episode in adolescence.

There appears to be mounting evidence that adverse childhood life events play a role in the development of bipolar disorder and impact the course of the illness. Again the mechanisms by which they contribute have yet to be fully understood. Psychological interventions cannot reverse these adverse events, but by understanding how they are relevant to the development of bipolar disorder we can use this knowledge to further develop psychological therapies. Given the wide prevalence of childhood traumatic

events, specifically abuse, in bipolar disorder patients, the use of specific psychotherapies targeting recovery from abuse should be investigated.

2.6 The interpersonal context

2.6.1 Social Networks and Social Support

This section aims to come to grips with the far-reaching concept of social networks and social support in relation to its connection with mental health, and furthermore to make them relevant for this study's background. Exhaustive examples of social support can be found in: Vaux (1988), Angermayer und Klusmann (1989), Sarason et al. (1990), Röhrle (1994) and Brugha (1995).

Social support is, in itself, not a new concept, whose beginnings can be traced back to a specific point in history. It would be more appropriate to compare it with the term "parenthood" as it is inherent in human societies. Moreover a conclusive link between social support and psychological health has been established since early days.¹ However, since the 1970s and in the interests of social science research, social support has to some extent been moving conceptually in the focus of theoretical observations. It was originally brought into sociology to help to describe collective associations of individuals with a high level of interactivity. The conceptualisation of this complex in psychology first created the possibility to operationalise in a meaningful way problems which are dependent on environment and context; in other words to enable one to depart from a purely individual-centric perspective. Nevertheless, "Social Support" does not strictly

constitute a standardised concept, but rather is dependant on a variety of conditions. Investigations in the area of social support are accordingly heavily dependent on the respective form of operationalisation.

Numerous attempts to throw light on the link between an individual's social relationships and his general or psychological health have been made (Brown and Harris, 1989). Psychologically important efforts to integrate network and support concepts into a single theoretical framework can be found in John Bowlby's attachment theory – in the scope of a predominantly developmental psychological approach –, or in analyses of stress and social support (e.g. Brown & Harris, 1978). An instructive suggestion made within this context is that social support is more susceptible to possible interventions than the relevant aspects of stress themselves (Cassel 1974). This point of view has contributed enormously to the rise in research drives in the area of social support. Attempts to build psychological theories of social networks however come across as being very heterogeneous, and significant models of individual psychology end up being applied to a broader social context, without taking into consideration the limitations of such a simplification.

Under the concept of "Social Support", the subjective evaluation of recognised support or burden in social interactions is described primarily on a conceptual or psychological level, whereas the concept of the "Social Network" outlines the objective pattern of social relationships (Sommer & Fydrich, 1991). In this way patterns and roles of an individual's social surroundings can be fundamentally and conceptually differentiated. Moreover it is important to distinguish between different ways of how these features may

¹ In his early observations on psychological health, Burton (1621) established links between psychological well-being and social relations, and quoted here a number of ancient authors

affect someone: negative effects as a result of deprivation or social support patterns with negative impact; neutral, or not destructive, influences, and finally causes, which through pro-activity and change have a positive effect on health (Röhrle, 1994).

2.6.2 Conceptualisation of Social Support

Social support is, broadly speaking, comprised of two main parts, which come together to form a third part (Leavy, 1983).

- *Structure* – each concept of social support has to incorporate individual availability and the existence of attachment
- *Role* – which support roles are made available by relationships
- *Process* – how people make use of potential supportive relationships in their surroundings

Alternatively, social support can be broken down in to the following parts: a) general structural social network-resources, b) specific supportive behaviour and c) the subjective evaluation of experience with social support (Vaux, 1988).

The analysis of the structure of social support has to describe, on a fundamental level, how much support from willing people is available to somebody, and what role they play with regard to the person concerned. Social network analyses help to create the most comprehensive overview of a person's relationship structure and the make-up of individual social support. These should include all social liaisons of a person and the relationships inside these liaisons.

such as Plutarch and Tully.

Essential aspects of a network analysis are:

- The *size* of the social network – generally speaking, a large social network is considered to be a positive thing, as it is easier to reach somebody in larger networks, to share a burden with several people, and specific information or practical help is readily available.
- Frequency of contact with individual members of the network, *closeness* – the positive features of large networks previously mentioned probably correspond only to networks with minimal closeness, inside of which relatively few people know each other. At the same time, however, networks with a comparatively lower closeness place a higher demand on social competencies and can easily overload an individual.
- *Strength of attachment* between the individual network members – apart from purely structural features, the quality of individual relationships is an important feature in social networks. Emotional nearness, complexity and reciprocity are typical signs of a first-class, qualitative relationship.
- Degree of initiative or receptiveness among one another, *reciprocity* – the balance of supportive relationships plays an important role satisfying experienced social support. In this way, imbalanced relationships can prove to be extremely stressful, although they offer the desired form of support for the person concerned.²

Further morphological features of social networks to consider include:

- the relative similarity of people in a social network – *homogeny*

² This connection becomes clear in cognitive assessments of social support, in which the acceptability of experienced support depends heavily on perceived symmetry (Brewin, 1995).

- number of *clusters* in the social network that are definable
- the relationship between the social network and *external influences*.

According to Wills (1985) the most important roles of social support can be divided into the following areas:

- Support of self-confidence or *emotional support* – this form of support offers confirmation of a person's self-worth and can be linked with the concept of unreserved positive esteem (Rogers, 1961). Such support is usually sought after when doubts about one's own abilities or worth are experienced; in practice this means confiding personal problems or concerns, something that is commonly possible in close and sustained relationships. As a general rule these relationship roles are made available to partners, close friends or relatives. If this support is not present, the result can be emotional isolation.
- *Instrumental Help* – concepts such as concrete help, practical or material support are equally common. The actual provision of daily practical help is described.
- Sociability and *social interaction* – this support role covers experiences shared in social activities that mainly serve to give enjoyment or relaxation.

Emotional support, practical support and social interaction may be considered as the three most important support roles that are researched and discussed in connection with psychological health, social support and stress. Moreover they have been integrated into the majority of measuring instruments. In addition to these features, Wills (1985) describes two further roles of social support, namely informative and motivational support:

- *Informative support* – Information, advice and recommendations are provided through supportive behaviour. Examples of such information can offer an objective appraisal of a personal situation or problem. This help often stems from sources that also give emotional support, seeing as the two functions are closely linked. It is however of particular interest to persons with psychological problems who lack informative help and how they may be able to access some, even if the area of emotional support is removed or unsatisfactorily fulfilled.
- *Motivational support* – This feature is almost completely of a theoretical nature, as up to now there is virtually no empirical evidence that proves a decisive link between motivational help and psychological health. Nevertheless, Wills (1985) argues that the stress factors associated with psychological disturbance are mostly of a chronic nature. Support which manages to maintain positive expectations in constantly difficult circumstances can above all be of great importance when dealing with psychological disorders.

When the links between structural features of social networks and functional aspects of social support are considered, a remarkable mutual independence of both parts can be seen. This is true of the relationship between the size of social networks and the quality of support experienced, as well as for aspects such as closeness or complexity and the functional forms of social support (Vaux, 1988). It is therefore of corresponding importance that both concepts are considered independent of another. Their inclusion in investigations of possible interdependency should be done while respecting their relative self-reliance.

2.6.3 Areas and Resources of Social Support

The question of which areas of life each relationship should be assigned to is to be answered with great care. The importance of an area depends entirely on the respective stage of life; examples that can have lifelong significance may include family, friends and social activities.

Various studies have highlighted how people who have experience in a wide range of areas of life demonstrate a more stable psychological state of health than those whose experience is limited in just one or two restricted areas (Thoits, 1986). What is meant here by categorising areas of life is in fact the differing worlds of each subject, such as work, family, certain friends, etc. There is also evidence that the closeness of social networks plays an important supporting role when recuperating from certain psychological disorders (Greenblatt et al, 1982). Moreover, patients who have a comprehensive social network are less of a burden to their families, and therefore enjoy correspondingly better chances of recovery (Maurin & Boyd, 1990). In most of the cases concerned, a broad and scattered social network that does not have any ties between family and friends for example is more often associated with positive results. Similarly a situation in which the necessary support comes from a wide background of areas of life is more likely to meet the conditions for a scattered network, than if support comes from only one area. In this way, employment outside of the family home may for example offer protection against depression to women who are constantly under the influence of stress factors (Brown & Harris, 1978; Warr & Parry, 1982).

It is here that the question of whether men and women draw on support from different sources may be raised. There is enough empirical evidence to suggest that this may be

the case, although a proven systematic connection of social networks or support with differences caused by gender has not been established. Schuster et al. (1990) found that men draw on support primarily from their partners, whereas women tend to turn to a wider circle of family or friends – and mainly to other women. Cohen and Wills (1985) ascertained in a literature survey that women engage in another relationship style; they experience fulfilled relationships through close and intimate attachments, whereas men experience these supportive attachments more through activities of a social nature or with a specific purpose.

After considering the sources and areas from which social support is obtained, it is then important to take into account the accessibility of each significant attachment (Henderson et al., 1981). Suffice it to say that a potentially protective relationship is less effective when the person concerned cannot be reached in the desired form.

The subjective acceptability of the respective support role is also an important factor in the process of social support – that way it is not possible for every relationship in a social network to be taken on with the same degree of effort. Similarly, most people have a pretty clear notion of who can or should provide the corresponding support; relatives, for example, are often considered to be more appropriate sources of social support than perhaps friends or professional helpers. It is often the case that depressed women believe that only their mothers or partners can give them the emotional support they need. Even if such ideas are not clearly expressed or even consciously recognised by the persons concerned, they still have a critical influence on the processes of support – how they are perceived or offered determines how effective they are.

As a consequence, acceptance and accessibility stand side by side with the perceived suitability of social support received in two-way relationships. It is important to note in this context that most of the support gauges commonly used include subjectively perceived social support. This perception – or the discrepancy of perceived support and corresponding ideals and experience – is something that has essentially been derived from a link with psychological health (Brown et al., 1986, Henderson et al., 1981).

The table below summarises once again the addressed structural differences of social networks and social support, which are also relevant to the questions raised by this study:

Table 2.2: Main aspects of social support

Existence of core attachments: Parents, siblings, friends, life companions, colleagues, etc.
Components of social support: The availability and acceptance of core relationships and the satisfaction of social support's definitive features: Emotional Support – intimacy, trust; Practical Support – informative und motivational support; Social interaction.
Various sources of support: Family, circle of friends, work, different activities, etc.
Attributes of a Social Network: Size, closeness and other network qualities.

2.6.4 Models of Social Support

There is a whole host of individual findings that each describe the influence of social support on psychological health, and a multitude of action models that ought to make this

connection understandable. Nevertheless, it seems that such explanatory models and action plans remain largely ambiguous. Isolated results are thus to be considered together with the background of their models and conceptualisation and cannot be proclaimed as integrative results. It is here that, above all, the very definitions of social support and its theoretical background vary drastically, while the models of social support's effects are more uniform.

Two main underlying theories dealing with the effects of social support on psychological health have been evinced and thus become the focal point of all further discussions:

- (1) Through a *buffer effect*, social support has only an indirect association with psychological health, when in the presence of stress; in this view, social support has no effect on psychological well-being when severe stress factors are absent.
- (2) Social support has a direct effect on psychological health: low levels of influence from social support (e.g. a lack of social relationships) can be linked with poor psychological health, and may have a quasi-causal effect on the manifestation and development of psychological disturbances.

In the following section, the evidence concerning both models shall be briefly considered – two comprehensive study reviews go into detail about the people who have been analysed, and the extent to which social support was used (Cohen & Wills, 1985, and Alloway & Bebbington, 1987).

There are no consistent results for a buffer effect of social support under the influence of extreme stress factors. Most studies in this area deal largely with measures to do with general psychological health, particularly with regards to symptoms of depression and

anxiety disorders in random samples; buffer effects usually occur in the form of attaining intimacy, when in general a gauge of emotional support has been used. In thirteen studies discussed by Cohen and Wills (1985), eleven of them displayed significant buffer effects. The results were to be seen consistently when, first of all, the necessary methodological conditions were met – avoiding statistical artefacts caused by the inadequate operationalisation of variables or unsuitable statistical practices (see Veiel, 1987, 1988); secondly, the corresponding measuring procedures referred to the perceived availability of social support – studies, which pick out network characteristics or do not show any real perceived supporting roles -; and thirdly the supporting roles that have been considered to enhance the commonly-used coping mechanisms. As a rule, no buffer effects are to be seen when the quality of personal relationships and emotional support are raised, whereas the way in which most studies carry out measurements do not discount the impact other supporting roles have. Buffer effects do not cease to exist however when general operationalisations of social integration are used.

Studies that use multidimensional, structural characteristics of social support demonstrate largely consistent results, namely that social support can be proven to have a direct and beneficial effect on psychological well-being. In a similar way, integration into a comparatively large social network can also be shown to have a direct, positive effect on psychological health, all other factors being equal. However, support in this common form of a good social involvement does not necessarily prove to be useful in the face of huge stress factors (Cohen & Wills, 1985). This study's results may be attributable to the general effect of social networks on feelings of stability, personal self-esteem, or even on feelings of extreme isolation in small social networks. Therefore, positive integration does not as such have an influence on well-being that necessarily improves the ability to deal with stress factors. As evidence of this case, one can cite the low levels of

correlation between measures of social integration and supporting roles, as well as the fact that a large number of social liaisons have no buffer effect, even though a single, trusted person can be enough for such an effect (Wheaton, 1982; Vaux, 1988).

The buffer effects found here are not shown to be artefacts of confounding stress and supporting roles. Studies that confirm the buffer hypothesis do not show any correlation between support and stress measurements, whereas those studies that demonstrated a clear link between these factors did not confirm a buffer effect (Cohen & Wills, 1985). Furthermore, it can be considered confirmed that the individually perceived quality of social support constitutes an important role, when correctly operationalised, for the buffer hypothesis, rather than 'objective' network measurements or current social operations. These results are produced in connection with acute incidents as well as with regard to ongoing stress factors (Kessler & McLeod, 1985). Cutrona and Russel (1990) analysed a number of studies relating to the possible, stress-specific effectiveness of different supporting roles. In doing so, occurrences that are out of an individual's control such as unemployment, illness, financial difficulties etc were demonstrated as being beneficial, along with a large amount of emotional support such as social integration, appraisal and concrete support. In contrast, informative help, feedback related to action plans and emotional support are regarded as being helpful for incidents that can be influenced like personal crises, pregnancy, work stress etc.³

³ The difference between "controllable" and "uncontrollable" critical life events comes from the research techniques of the life event works by Brown and Harris (1978, 1989). In these works, "controllable" events are those that can be prevented or whose consequences for individuals can be mitigated. On the other hand "uncontrollable" happenings are supposedly not subject to an individual's influence, as the key agent of the event is another person or external factors have a considerable hand in the event.

However, these clear results can only be used up to a point. In reviews the different social support studies are simply listed and described, and as a result there is no unifying concept model in these evaluations. The consequence of this is that the heterogeneity of the individual results is reflected in a perpetual inconsistency of integrative concepts. Similarly, fundamental methodological differences between the discovered effects are simply assumed to be similar, rather than being standardised through statistical practices. This lack of consistency between main and buffer effects found in the different study reviews also led to an increase in the supposed impact of social support.

Over and above the presented main and buffer effects of social support, there is a set of further models which have been accepted as also showing the effect supportive behaviour has on psychological health. Nevertheless, only a simple overview of the different approaches should be given here. The starting points relevant in the context of this study, as well as certain process models on dysfunction are to be commented on with more detail in connection with the dysfunctional images discussed in this work.

Early enhancements of the general concept are largely due to the move away from purely quantitative models towards a cognitive design i.e. focusing on the subjective appraisal of social support (e.g. Cutrona & Russel, 1987). Accordingly, it is now solely the perception of social support that constitutes the most operationalised gauge of support. By means of this, it demonstrates its clear impact on psychological health. Another aspect of social networks, which has a conducive effect, directly or indirectly, on psychological health can be mentioned here in this context; the role of stabilising a person's feeling of self-worth. It is assumed here that a person draws his or her identity and worth from how, they presume, important persons in their life perceive them (Heller et al., 1986), whereby the stability of identity and self-worth is dependant on integration into a social network. This

basis on social interaction also comprises aspects that have a positive effect on health, such as the exchange of informal help and information, as well as the company of others in social activities.

Altogether, the fundamental models of the effect of social support illustrated have changed and been in many cases complemented by the common understanding of the concept of social support. Unidirectional, semi-causal models of the effect of social support or stress factors on physical, as well as psychological health have been revised and updated in favour of interdependent impact mechanisms. In this way the impact of social support is more exactly divided up according to causal and time factors. In doing this it can be said that social support shows an advantage even before a stress factor appears (Champion, 1990), or it can be strengthened, even activated through the effects of stress factors (Lin, 1986). A number of additional factors also play a decisive role in the individual assessment of social support. As such, specific effects result from the consideration of the interdependency of social support and the influence of stress factors. These effects do not only just show dependence on the type of stress factor (Cohen & Wills, 1985), but also show curvilinear relationships, in the sense that stress factors can have a catalytic effect on social support, as well as a destabilising one if they are particularly fierce (e.g. Tezloff & Barrera, 1987).

Factor analysis can equally demonstrate the multidimensional quality of social support. McCormick et al. (1987) showed that the size of the social networks, frequency of support and satisfaction are due to varying factors. In a further factor analysis Sommer and Fydrich (1989) describe the emotional and practical operating spheres of social support, as well as social integration and social pressure factors.

All in all, the heterogeneity of the concept shows that it is necessary to comprehensively take the different features of social support into consideration when analysing possible influences.

2.6.5 Positive and negative aspects of social support

In order to better understand the processes that contribute to the effectiveness of social support, additional criteria have to be considered. When picking out support factors, the irksome aspects of social attachments were mostly disregarded – the negative effects of social stress factors, as well as negative and burdensome attachments were paid inadequate attention. Moreover, possible confounding factors or interactions between these two aspects of social interaction were in general insufficiently considered.⁴ According to Rook (1992), these negative or problematic attachments are identified in the following way: the treatment of persons in a social network that causes suffering to the individual concerned. This also includes well-meant assistance that fails by meeting with rejection, conducting damaging behaviour or that is simply over productive. Here the perceived sources of social support are often the same as for social stresses. In addition, the subjective perception of support can be affected by potential stress factors, whereby lower levels of support occur not from a lack social support, but as a consequence of differently associated stress factors. This is how the potential availability of support can be directly limited by stress factors. Conversely, the existence of appropriate support can pre-empt the appearance of certain stress factors (Champion, 1990).

⁴ The largely heterogeneous findings on positive and negative aspects of social support can in part be traced back to the fact that both parts were seldom picked out together. It was more often the case that more conducive parts of social support, as well as unrelated negative contingents of social attachment were considered (e.g. Sommer & Fydrich, 1989; Schuster et al., 1990; Rook, 1990).

Schuster et al. (1990) ascertained that it is often the closest and most familiar persons who can also be the most probable source of extreme pressure. Sommer and Fydrich (1989) found significant correlation coefficients of $r = -.40$ between perceived social burden and different supporting roles. Social burden, or pressure, in this context means relationships that are felt to be a strain i.e. not missing social support but attachments that are affected by "*Criticism, rejection, excessive demands and lack of distance*" (Sommer & Fydrich, 1989, p. 163). In the life-event research it emerged that particularly in cases of depression, significant interpersonal stress factors are often to be seen before a bout of depression sets in (Bebbington et al., 1988).

The processes of negative and positive aspects of social support in relation to psychological health are both presumably equally complex. Even negative aspects, such as well-meant pushy or encouraging behaviour, come across in direct and indirect forms, according to all predictions. Such research efforts that observed the positive and negative sides of social relationships show that negative effects have a significantly stronger influence on psychological well-being, although they occur more seldom (Schuster et al., 1990; Rook, 1990). At the same time, two strategies can be determined: one incorporates the comparison of the effects of positive and negative exchange processes regarding psychological health (e.g. Fiore et al., 1983; Rook, 1984), whereas the other strategy compares the stress-relieving influence of social support with the stress-boosting effects of social conflict and negative interactions. With this last strategy in mind, Okun et al. (1990) examined the role of negative relationships with regards to the hindrance or deterioration of the ability to adjust to acute or chronic stress factors. The participants of this study were 110 older persons receiving outpatient treatment. Aside from the perceived stress factors of the last six months, negative social attachments and

psychological strain were ascertained through questionnaires and interviews. Particular attention was paid to ensuring that the social factors were in no way linked to negative relationships in social networks. Regression analyses, in which the effects of physical health and socio-demographic differences were monitored, demonstrated a significant core effect of negative social attachments concerning psychological health, but not for positive relationships. Moreover, the study by Okun et al. (1990), and the similar investigation by Finch et al. (1989) displayed an independence of this effect from the stress factors type and strength. Those who experience little stress, as well those who are exposed to heavy strains both show a connection between negative interactions and psychological pressure.

In this context it is the reciprocity of social relationships that can be felt to be a great burden when there is an imbalanced two-way action of support (e.g. Buunk et al. 1993). Such an imbalance often occurs in groups that are not in a good position to maintain reciprocal relationships. Examples of this are groups with a low income or persons with a long history of psychiatric institutionalisation, as they are often wholly dependant on the one-sided support of others. Concerning mutual supporting benefits, Antonucci & Jackson (1990) found that people from a handicapped group tried to maintain balanced relationships in their social networks, and that a significant correlation existed between the perception of a lack of reciprocity and psychological strain. Antonucci & Jackson (1990) were able to confirm these findings by comparing different groups of varying nationalities and ethnic backgrounds of 6400 participants. Both forms of imbalanced relationships - whether they in their own view give more support than they receive or whether they receive more support than they give - are associated with lower levels of life satisfaction as compared with groups with balanced ties.

This link between low socio-economic status and an increased risk of psychological disorders can in part be traced back to the high proportion of burdensome relationships. It can also be attributed to the fact that the social situation of low income groups restricts the choice in creating a social network and in conducting individual interactions, as well as allowing only limited access to poor and burdened networks (Brown & Harris, 1978; Belle, 1982; Rook, 1987).

As Fiore et al. (1983) demonstrate, it is above all depressed patients who often have unrealistically high expectations of their social networks, and thus are correspondingly more often disappointed. Parry and Shapiro (1986) illustrated that depressed patients overemphasise the negative aspects of relationships due to their own low self-confidence. Moreover they negatively speculate that they will never be able to return the support given, which in turn blocks their ability to muster support or to accept support when it is offered. In a re-analysis of two studies by the Royal Holloway and Bedford College Teams, Harris (1992) found that, alongside a host of other non-supportive or negative behavioural influences of relatives on the relapse rate of depressed women, patients who had received a lot of support during an initial bout of depression from their partner or family members suffered even stronger relapse when this support ceased after time, or when they were disappointed from support given later.

A further aspect of negative relationships is experienced criticism or over-involvement, and how the stand in connection with the expressed emotion concept. The link between negative interactions in this sense and possible relapses has been repeatedly confirmed, particularly for psychiatric groups (for an overview: Kuipers & Bebbington, 1988).

2.6.6 Social Support and Psychological Disorders

The awkwardness of the dependence of different results on the process model used or on the methodological framework of the study has been noted time and again in summaries and reviews of social support literature (e.g. Henderson, 1980; Röhrle, 1994). When trying to illustrate this area, it is not easy to avoid these difficulties. This is because in the context of social support and psychological health there is no commonly accepted research paradigm or theoretical model. As a result, it is difficult to compare many findings.

In general, the proof of a link between social support and future clinical states is based largely on studies that concentrate on individual groups that already suffer from a psychological disorder, rather than on those that concentrate on the illness from the beginning (McLeod et al., 1992). Furthermore the manifestation of the factors concerned differs from those that may influence recovery (Brugha et al., 1993). While the improvement and course particularly of depression disorders can be predicted by looking at how the perceived lack of social support during the period of illness develops (Jablensky et al., 1992), there is an increasing amount of evidence that suggests that other aspects of social interactions also have an influence on the subsequent development of clinical relevant symptoms (Brugha et al., 1990).

In addition, some research findings suggest that there are systematic differences in how social support impacts on varying diagnostic groups, in which a specific process model is partly assumed. Thus a multitude of studies attempt to provide evidence for differing structures of social support in various subgroups of depressive illnesses. The results show comparably deficient social networks for both groups: patients with “neurotic”

depression and retarded depression (Brugha et al., 1987a, b). However, the results of a four-month follow-up study showed a markedly more positive link between social support and recovery in the group of patients with “neurotic” depression. This link was not confirmed for the second group (Brugha et al., 1987b). Romans and McPherson (1992) discovered that the social networks of bipolar depressive patients became more impoverished as an increasing number of manic phases followed. Brugha et al. (1993) confirmed these findings by demonstrating that in a comparison between long-term and acute psychiatric disorders, the social networks in the first group were restricted. Moreover, the networks of schizophrenic patients were not smaller than those of patients with other chronic disorders e.g. with recurring affective disorders.

All in all, there is still no clearly structured and comprehensive pattern as to how certain types of social support work in specific cases of disorders. Previous isolated attempts at differentiating forms of social support display a heterogeneous picture on the whole, and yielded no consistent results.

2.6.7 Moderating influences of Social Support

In short, it can be presumed that a multitude of individual determinants that are specific to development and sensitive to the surroundings influence the form, structure and development of individual social support systems.

According to Brewin (1995), there are three important cognitive processes that influence the perception and use of social support: firstly, personal or inner belief of one's own social competence; secondly, the role of social comparisons, and thirdly, the effect of a feeling of self-worth.

Thus socially-timid and lonely people tend to ascribe interpersonal failures to supposedly stable internal factors. This form of association leads on to lower self-esteem and self-efficiency. This in turn disinclines the person yet more to form and maintain social contacts (Leary et al., 1986). This link between cognitive processes and the subjective assessment of social support becomes clear in connection with depressogenic cognition errors (Beck et al., 1979). Thus depressed individuals evaluate others modelled on their perceived self-worth. The relevant social information can only be accepted in a way that one's own self-perception allows (Sarason et al., 1991). According to Kuiper and Derry (1982), depressed persons tend to value themselves less and less in social contexts. This negative view of oneself is often linked to the loss of significant attachment figures. All in all, depressed people assess their potential social support in a negative way, which makes the real lack of help seem particularly pathogenic (Lakey & Cassedy, 1990). In contrast, chronic psychiatric patients tend more to over-estimate their potential social support (Klein et al., 1987), which however does not apply to the area of close confidants or role models (Barrera et al., 1985).

A further key cognitive process is described definitively by Goffman (1968): passed on through social rejection and difficulties experienced, psychological illness can often be traced back to a self-imposed social isolation. Alternatively they try to conceal parts of particular self-disclosing behaviours, which usually makes social interaction impossible. In particular it is how the close social spheres respond to the behaviour of the person concerned; negative reactions strengthen this behaviour. However, other social experiences make a significant contribution to self-stigmatisation and social isolation. These persons often try to avoid relationships with "normal" people i.e. psychologically normal people, and search increasingly for contact with people in a similar situation.

These process may well play a part in the fact that depressed people have smaller social networks. They perceive themselves to be different from others. Brewin and Furnham (1986) think that these beliefs lead depressed people to avoid other people, thereby shutting themselves off from advice and normative information that could have a corrective effect on this behaviour.⁵

From this argument, it becomes clear that social processes of comparison are closely linked with the formation and maintaining of an individual's self-esteem. By conducting interviews with inconspicuous people, Folkman et al. (1986) investigated which coping strategies they used when confronted with stressful events. The findings showed that a threat to self-esteem is associated with reinforced feelings of flight and avoidance and less supporting behaviour. In another analysis this data, Dunkel-Schetter et al. (1987) confirmed a consistent inverse correlation between a perceived threat to self-esteem with support measures. This includes the amount of information received, practical help and emotional support, as well as the number of sources of social support available.

Studies by Andrews & Brown (1988) and Andrews & Brewin (1990) show how useful these cognitively mediated psycho-social models are, particularly when treating depressed patients. Suboptimal trust of close attachment figures due to the already anticipated negative expectations and the turning of criticism into self-guilt are destructive components of depression. The above mentioned models can be used to confront these destructive elements in a therapeutical context to reduce unfounded mistrust and to support the use of more resources in a wider social network.

⁵ There is a number of empirical studies that show the connection between a cognitive style of attribution or certain beliefs and avoidance behaviour of psychologically ill persons (Snyder

In a longitudinal study with working-class women from Islington, Brown et al. (1986, 1990) examined the influences conveyed from an individual's self-esteem. They noticed that a negative self-evaluation has an important hand in creating an additional vulnerability factor when a depressive episode occurs due to a negative experience. There are also a number of studies that link other personality factors, such as sociability or extroversion with supportive social networks. Thus Sarason et al. (1983) found that how extrovert a woman is can be linked to the size of her network, and satisfaction with help received to low levels of neurosis.

The symptoms of psychological disorders, particularly those that commonly have long-lasting negative consequences, probably have a negative effect on the support of others e.g. from family members. With this in mind, disorders such as depression, phobic anxiety disorders or paranoia represent particular examples how social contacts reduce directly as a result of such disorders. Barrera (1986) names about fifty studies that show a negative link between psychological suffering and support factors, above all "social integration" and "perceived support". This effect can be divided into several levels. Apart from the direct effect that suffering initially engenders enhanced support, a kind of wear and tear on supportive relationships, particularly in chronic cases, can be seen (Chestler & Barbarin, 1984). Supportive behaviour is also not just "consumed", but also compromises close relationships within an existing social network, as principles of reciprocity and trust are undermined. Thus chronically ill patients often have networks that consist of a few, non-reciprocal and informally linked relationships (Cohen & Sokolovsky, 1978). Consequently, this noticeable drop in support in social networks also changes the individual evaluation by the person concerned regarding social support

& Ingram, 1983 and Brewin et al., 1989).

received. They often become unhappy with the perceived support and feel less respected and cared for than others.

Therefore it is necessary to take into consideration the logical-causal difficulties of association between the psychological disorders and the patterns of social support discussed above.

From a theoretical point of view, social support has to be understood in a further, developmental psychological context. A study by Skolnic (1986) examined the link between early mother-child relations and the quality of social relationships over an entire life span, in a sample consisting of both male and female Americans. However, the findings of this study are not consistent for the purposes of attachment theory. Poor mother-child attachments in the first two years of life don't necessarily lead to impoverished childhood or adolescent relationships. An interesting result was the apparent importance of childhood peer attachment. The quality of these peer attachments was closely associated with the development of satisfactory relationships in adolescent and adult years.

Similarly, when the relationship between stress and social support is contemplated in a development-related context, and correspondingly not restricted to the conditions that make up the manifestation of a psychological disorder, there is the probability that positive social support works as a primary protector against the appearance of huge stress factors. A re-analysis of data from studies on critical life experiences and depression suggest that a lack of emotional support is linked to an accumulation of negative life experiences (Champion, 1990). It is equally striking from a developmental, psychological perspective that in more advance years a noticeable increase in the positive

perception of social support can be observed (Lam & Power, 1991), as well a fall in negative life experiences (Henderson et al., 1981).

It cannot be conclusively deduced here how social support can be meaningfully analysed in a development-oriented framework. However this attempt to understand a number of factors is important in the scope of the observation of interaction process of social support and psychological health. Firstly, perspective contributes to the clarification of a person's intrinsic qualities, such as personality variables, self-image, coping and social capabilities. Secondly in broader sense, developmentally relevant influences shape the experiences with the social surroundings – the presence or absence of confidants, the quality of care that is received from this person, as well as experiences of rejection, loss, cultural valences and the form of sociability experienced.

Inter-personal therapy targets social support as a key area, in particular the importance of having adequate social networks. By definition, social support refers to the perceived availability and quality of close relationships (Cohen et al., 2004). High-quality support networks are thought to act as buffers against negative life events in times of stress and poor interpersonal relations can in turn produce stress (Frank et al., 1997). However despite their greater need for support, individuals with bipolar disorder are shown to have comparatively little social support compared to individuals without the disorder (Wilkins, 2004).

The interpretation of social support is highly contextual and idiosyncratic to the individual (Morriss, 2006). Adequate social support can come from a small number of core friends or family members, from a network of people, or from different sources depending on the problem. It usually takes the form of both practical and emotional support. Findings

from a study of befriending as an intervention for chronic depression suggest that the person's perception of the availability of social support may be of greater importance than the actual support itself (Harris et al., 1999).

Several studies have examined the relationship between social support and the aetiology and course of the illness. O'Connell et al. (1991) found a strong correlation between poorer outcome in the course of bipolar disorder and low levels of social support. In a retrospective study by Stefos et al. (1996) social support as well as other psychosocial variables, but not clinical variables such as age or illness duration, predicted the occurrence of a major affective episode. Johnson et al. (2003) rated 94 stabilized patients using two social support questionnaires. Patients with low social support were more likely to relapse over a 1-year prospective follow-up period. In addition, significantly lower social support was found in those who made a partial recovery compared to those who made a full recovery. Having a partner was also found to contribute to interepisodic recovery. Cohen et al. (2004) also found an effect of social support on relapse, reporting that lower levels of perceived support significantly contributed to the recurrence prospectively over 1 year. However, other studies have not found support for such a relationship. Kulhara et al. (1999) found that social support and life event related stress were psychosocial correlates of lithium response. Staner et al. (1997) found that social support was not a predictor of new affective episodes.

Polarity specific effects have been found in the relationship between social support and course of bipolar disorder. Johnson et al. (1999) found that as well as high social support being associated with quicker recovery from mood episodes, it is associated with a decrease in the likelihood of depression, but not mania. Other studies have supported this finding suggesting a polarity effect similar to that discussed with EE and highlights the

possible utility of different interventions at different times (Johnson et al., 2000). However, this polarity effect is not consistently found (Johnson et al., 2003).

Social support has also been shown to affect psychosocial outcome in bipolar disorder. Hammen et al. (2000) found that presence of a good quality supportive relationship was the strongest predictor of work functioning, more so than clinical factors. Wilkins (2004) also found that likelihood of employment was significantly greater for bipolar individuals with higher levels of social support. It would appear from these findings that having (or perceiving to have) a good source of social support may help to reduce the negative impact that the disorder has on work functioning.

A number of possible explanations seek to elucidate the relationship between low social support and relapse. One theory is that lack of social support leads to a vulnerability to relapse, through a lesser threshold for stressors. However, Cohen et al. (2004) found that there was no moderating impact of social support on stress. Equally another possibility is that the disorder itself leads to the inability to interact in an adaptive fashion. Goldstein et al. (2006) examined social skills deficits among a small group of adolescents with bipolar disorder and controls. They found that the bipolar group displayed significantly more deficits than the control group in terms of social skills performance, but the two groups did not differ significantly with regards to social skills knowledge. This suggests that bipolar adolescents are able to understand information regarding interpersonal tasks and social rules similarly to healthy controls, yet somehow lack the ability to exert control over their behaviour in order to achieve interpersonal aims. This could, in part, also explain the lower levels of social support found in bipolar patients compared to controls. This is pertinent to psychological intervention aimed at improving interpersonal relations.

Other possible explanations of the relationship between low social support and relapse include personality factors underlying the course of the illness and the degree of social interaction, or patients underestimating their social support (Johnson et al., 2003). Social support may be a significant correlate of response to medication, meaning that people with good social support are likely to comply with their medication regime, as in Kulhara et al.'s (1997) study. A related concept that has been studied is social adjustment. In one study of predictors of recurrence, social adjustment was found to be the strongest risk factor in the prediction of new affective episodes, whereas social support was not a significant risk factor (Staner et al., 1997). However, this was a relatively small sample and unipolar and bipolar patients were not separated for analysis.

Social support can be examined from a more systemic view by including research into relationships from relative's points of view. Spouses of patients with affective disorder in remission have been found to score their marital relationship lower on qualities such as unity and affection than spouses of controls (Levkovitz et al., 2000). An older study found that couples with one partner with bipolar disorder showed higher levels of marital disharmony than control couples (Hoover & Fitzgerald, 1981). Furthermore, the patients reported significantly more conflict than their spouses, which may have implications for perceptions of conflict and support. Increased rates of divorce and separation have also been noted in bipolar disorder (Suppes et al., 2001; Wilkins, 2004).

Measures of social support and social adjustment have been found to have relationships with other key clinical variables. Bauwens et al. (1991) found that in bipolar individuals, who scored lower on measures of social adjustment than controls, scores were partly related to the number of lifetime episodes and current residual symptoms. Romans &

McPherson (1992) found that euthymic participants reported less social support than a control group. Social interaction decreased as a function of age and duration of illness and mania had a negative effect on relationships compared to depressive episodes. However, other researchers have not found support for the relationship of social support and age and duration of illness (Stefos et al., 1996; Johnston et al., 2003).

A methodological point to be debated is whether social support should be measured by perception of the patient, or by a more objective measure. Many of the assessment tools used in the above studies lack an objective measure of support availability and quality. Beyer et al. (2003) found that both older and younger patients with bipolar disorder perceive their social support to be inadequate. For the older patients, there was no difference in the number of interactions or amount of support that they received. However, in younger patients there was a decrease in number of social interactions. Given the suggestion that the person's perception of the availability of social support may be of greater importance than the actual support itself (Harris et al., 1999), there may be merit in assessing the discrepancy between the two, and perhaps using this as a focus for psychosocial intervention.

One last note of caution is that it is likely that measures of social support overlap with measures of self-esteem. Johnson et al. (2000) found that self-esteem was the most important predictor of change in depression across a 6 month period, but acknowledge that there may be an overlap in the concepts of self-esteem and social support.

2.6.8 Family factors

It has long been recognised that bipolar disorder is characteristic by substantial impairment in family functioning (FF). Families of patients in acute episode have been shown to score more poorly on FF ratings than those of normal controls (Friedmann et al., 1997). Specific aspects of family functioning which impact the course of illness include expressed emotion and negative family affective style (Miklowitz et al., 1988). Expressed emotion will be examined as a factor in its own right in the next section.

The McMaster Model of Family Functioning (MMFF; Ryan et al., 2005) permits systematic evaluation of patterns of family functioning across multiple domains (Weinstock et al., 2006). The model assesses family functioning across the dimensions of: problem solving; communication; roles; affective responsiveness; affective involvement; behaviour control; and overall general functioning. Lauren Weinstock and colleagues (2006) used this model to attempt to reveal unique patterns of family impairment associated with bipolar and unipolar disorders. The overall results showed that the two groups were more similar than different in their patterns of family functioning. Both groups showed a significant improvement in FF from acute episode to recovery. Despite this improvement they also found that mean scores at recovery continued to range from fair to poor in both groups. This finding suggests that whilst FF is to some extent state-dependent, it is still present to a sizable degree during recovery suggesting there is potential for family work during all phases of bipolar illness.

Uebelacker et al. (2006) examined whether global family functioning was associated with mood episodes concurrently and in the subsequent 3 months. Using the Family Assessment Device (FAD, Epstein et al., 1983) and the McMaster Clinical Rating Scale

(MCRS, Miller et al., 1994) to measure family functioning, they found a relationship between concurrent mood state and FF. This highlights that family therapy could be a vital form of intervention. However the study found no association between FF and change in episode status. It is perhaps the case that only specific aspects of family function (such as EE) are associated with course of illness. It is also of note that the patients enrolled in this study were part of a family-treatment trial (Uebelacker et al., 2004, with two thirds of the sample receiving FFT. However treatment group was not found to moderate the association between family functioning and episode status.

2.6.9 Expressed Emotion

Within the comprehensive discussion of the concept of social support, expressed emotion is often referred to as one of the most qualitative aspects of social attachment, particularly in connection with how psychological and schizophrenic disorders take their course. The term “Expressed Emotion” (EE) is meant to describe the mutual emotional reactions and behavioural traits of family members in the same household. In this context, the marked level of negatively affected attitudes of family members towards patients, such as a high level of criticism, dismissive demeanour or over-involvedness, turned out to dramatically increase the danger of having a relapse.

The concept was originally introduced by Brown & colleagues (1958, 1962, 1972). They observed that schizophrenic patients who returned to live with their parents or partner after being discharged were more likely to suffer a relapse than those who lived with siblings or had little contact to their relatives. Furthermore they were convinced that social factors that play an important role in causing relapses for schizophrenics must also be present in day-to-day family interactions.

Social interactions can be interfering, intrusive or extremely critical. The effects of such relationship models were examined in connection with relapses of schizophrenic patients and extent of the relatives' expressed emotion (Vaughan & Leff, 1976). Three important factors emerged that help to predict relapses:

- Critical comments
- Hostility
- Emotional over-involvement

Vaughan and Leff (1976) showed that high levels of EE represent a better predictor of schizophrenic relapses than negative compliance of medication. This is true for patients, who come from a family environment, in which weekly contact between family members exceeds 35 hours. This is also virtually the case for depression too, in which high relapse rates occur in patients who are exposed to family surroundings with a marked level of EE, although here the amount of critical comments proved to be pivotal. For depressed people, a low level of direct contact was associated with a negative outcome, whereas less direct contact acts as a protective barrier for schizophrenics.

A number of studies confirm how EE can be seen as a causal variable of relapses in schizophrenic disorders (Kuipers & Bebbington, 1988), regardless of culture and language (Kuipers, 1992).

2.6.10 Operationalisation of Expressed Emotion

Expressed emotion is often determined by assessing the recording of semi-structured interviews with relatives, the Camberwell Family Interview (CFI). It allows one to pick out the subjective attitudes of feelings of the relatives towards the patient. The interview covers the initial phase of problem development, such as illness development, disputes, shared activities etc, focusing on the time frame of a month before the interview and includes other aspects of the relationship, such as huffishness and inter-subjective tensions. The interviewer asks particularly about specific symptoms and coping strategies. A timetable of a typical week is also created, in order to get an impression of how and how long contact occurs for. The final rating will be made according to what is on tape. This comprises not just statements made but also linguistic aspects such as voice pitch, strength and intonation, as well as observations to do with non-verbal aspects such as gestures and facial expressions. In doing this, it is possible to assess emotional viewpoints without drawing on the relevant content. This EE procedure uses the individual behaviour of a relative under the artificial conditions of the CFI to work out in advance the probability of future relapses by patients living with these relatives. This is possible, according to general opinion, because the significant and constant features seen in the interview illustrate the interaction between patients and relatives, or the coping strategies of relatives.

Aside from the time-intensive CFI, it is also possible to measure EE by using the Five-Minutes Speech Sample –FMSS (Magenau et al., 1986). In this method, the original, complete, semi-structured interview is replaced by a five minute verbal appraisal of the relatives – how they come to terms with the patient and what sort of person he or she generally is. In FMSS, only the two original dimensions of EE are considered: emotional

over-engagement and criticism. The concurring assessments of EE by the CFI and the FMSS are judged well in corresponding validation studies. In cases where there was a lack of congruity, the findings from the FMSS tended to underestimate the degree of EE (Magenau et al., 1986; Leeb et al., 1993). Since then there are now self-report scales to bring together analogous EE factors about the assessment of patients themselves (Kazarian et al., 1990; Shields et al., 1992). These managed a satisfactory correlation with the CFI in validation studies, and above all made a predictive evaluation of the frequency of relapses, comparable to the other measures of EE, possible (Hooley & Teasdale, 1989; Franks et al., 1992; Cole & Kazarian, 1993; Shields et al., 1994).

Investigations, which consider EE as the causal variable of relapses in schizophrenic disorders, are based largely on a comparable design. As a rule, a group of patients are supervised for specific period time after having recovered from an active phase. Correspondingly, an interview with the relatives is carried out at the time when the patient is discharged (CFI), in order to record the original degree of expressed emotion. The patients are then re-examined after nine months or a year for signs of possible relapses. In some cases this is determined by how many relevant symptoms can be seen and how strong they are, or in other cases simply by the re-admission to a psychiatric institution. There are several methodical flaws to be noted in a number of these studies. Firstly, most of illness periods were not checked properly. Secondly, the criteria often drawn up to measure "re-hospitalisation" or "relapses" are not operationalised in a uniform way, which may restrict how much they can be compared. However, the relative robustness of the findings, and the prognostic value of the EE variables speak against these objections. A further methodological point of criticism is to be seen in the prevalent dichotomisation of the EE variables.

It was relatively early on that discussions about the genesis of the recorded behaviour of close relatives (Expressed Emotion) became more pronounced. Thus it may be that EE is only predictive because relatives react to patients in specific ways that have a negative prognosis for other reasons. In the first exemplary-planned study of EE (Brown et al., 1972) the influence of positive characteristics of symptoms was monitored. Nevertheless, EE held onto its predictive nature. Birchwood and Smith (1987) argued that a high level of EE and the associated behaviour of several close relatives are developed as a means of coping with living together with a schizophrenic person. They give reasons for their statements, insofar that high levels of EE in a family member, who experience the illness of a relative for the first time (in contrast to those who experience several repeated illnesses), are rarer. Very little can be said against this model when it says that different ways of coping are developed through the course of living with a psychologically ill relative, as it would be difficult to imagine how EE could develop except from in the interactions between a patient with their family. This does not preclude however the fact that these ways of coping have a negative influence on the further process of recovery.

In table 2.3 a number of research studies have been put together that suggest a relationship between high expressed emotion and certain other psycho-social aspects and characteristics of families or comparable cohabitation. Some are strictly speaking not closely linked to another, although they can nevertheless appear together in isolated families. The compilation is structured according to three points of view: characteristics of relatives with high levels of EE, features of patients who live with high EE relatives, and signs of interaction patterns between these relatives and the patients.

Table 2.3: Studies into various behaviour and attitude characteristics of families with high levels of EE

<p>Family relatives:</p> <p>Direct criticism (Rutter & Brown, 1966)</p> <p>Difficult family atmosphere to appraise (MacCarthy et al., 1986)</p> <p>Anxieties and fears (Greenley, 1986)</p> <p>Negative affect styles (Miklowitz et al., 1989)</p> <p>Poor listening (Kuipers et al., 1983)</p> <p>Ineffective ways of coping (Bledin et al., 1990)</p> <p>Avoidance, ignorance and resignation (Birchwood & Cochrane, 1990)</p> <p>non illness related attributes (Brewin et al., 1991)</p> <p>Attribution of bad courses (Brewin et al., 1991)</p> <p>Patients:</p> <p>Critical towards relatives (Strachran et al., 1989)</p> <p>Little autonomy (Strachran et al., 1989)</p> <p>Small social competences (Barrowclough & Tarrier, 1990)</p> <p>Interaction::</p> <p>Negative family model of escalation (Hooley, 1986)</p> <p>Negative emotional environment (Hubschmitt & Zemp, 1989)</p> <p>Conflict-ready structures (Hubschmitt & Zemp, 1989)</p> <p>Rigid model of integration (Hubschmitt & Zemp, 1989)</p>

Over the past few years, research into EE has experienced an interesting enlargement by taking into consideration EE in other relative contact persons, such as professional care workers. Moore et al. (1992a, 1992b) found that health care workers show an above-average level of EE with regards to their patients. In an interaction study of care workers and patients (Moore & Kuipers, 1992) it was shown that workers with high levels of EE usually made fewer supportive comments, whereas care workers with lower levels were more in the position to get away from their own negative feelings (should there be any) and thus not to concentrate too much on the negative behaviour of their patients. This then allowed constructive interactions that aided recovery.

Although certain behaviour patterns or disorder-specific characteristics of patients represent a burden for family relatives, and thus to suboptimal coping strategies (i.e. high degree of EE), it is plausible that these reactions do not necessarily follow as possible forms of interaction. Bertrando et al. (1992) have shown that there is no consistent link between the relatives' EE and the EE of the corresponding care workers of a patient. It can thus safely be said that model of behaviour patterns typical of high EE and common relapses does not represent a linear causality, but rather a spiral characterised by different interactions. Once this vicious circle has set in, EE represents a remarkably stable feature in the families. MacCreadie et al. (1993) found a notable stability of EE measures over a period of five years. In both studies, there was only a small number of relatives in which EE values fluctuated between high and low levels. These variations point the finger however to a group of family members that show typical EE reactions particularly when under stress. The relapse rate in this group of fluctuating levels of EE is interestingly enough similar to the group with consistently high levels of EE, whereas both groups differ significantly in their relapse rates to the group with consistently low levels of EE.

Expressed emotion addresses the quality and the style of interactions in family units that are necessarily complex. Living together with relatives who have high levels of EE presumably poses a constant stress factor for psychiatric patients. In this sense, relatives with low levels of EE are not just neutral but are associated with high levels of supportive behaviour and a positive attitude (Hubschmitt & Zemp, 1989) and are capable of defusing conflicts rather than letting them escalate (Hooley, 1986). Social surroundings that are labelled as having low EE lack not only negative aspects, but also have the potential to positively influence the patients. Similarly, frequent contact with low EE relatives has a protective effect on patients who are not being treated with medication i.e. those who

are particularly vulnerable (Tarrier et al., 1988). In this context, the convalescent effect of social support and the negative effect of social burden and stress factors presumably overlap.⁶ Thus, with simultaneously low levels of EE and high levels of social support, the relapse rate fell from 22% to 9%. This form of 'non-intervention' social support offers help as a reaction to the needs of the patient, without the patient feeling pressurised not to refuse. This form of support has a negative correlation with 'emotional over-involvement' (Brown et al., 1972; also Kavanagh, 1992).

An additional form of construct validation of the concept of EE can be found in studies that incorporate psycho-physiological variables in order to support the effect of high EE surroundings as constant stress factors (e.g. Tarrier et al., 1979; Sturgeon et al., 1981). In a replicated study, Tarrier (1989) found different autonomic forms of agitation in active interactions between schizophrenic patients and high versus low EE attachment figures. While patients with low EE relatives adapted quickly to interactions with them, patients exposed to high levels of EE demonstrated a higher level of skin conductance, as well as increased blood pressure. High levels of EE in a close attachment figure act as direct and constant stress factors when regularly exposed to it.

2.6.11 Expressed Emotion and Psychological Health

Traditionally, EE was mainly examined in connection with the probability of relapses in schizophrenics. Subsequently it was considered to reliably predict relapses, as well as a meaningful starting-point for preventative intervention against relapses. However the common framework of the EE concept, in which psycho-social strain factors (mainly

⁶ Even in the original study by Brown et al. (1958) comparable relapse rates were to be seen in patients who lived alone, as well as in patients who live with high levels of EE. This led

forms of interaction in the patients' families) are contemplated in connection with the developing of psychological disorders, suggests that these factors play an equally important role for other disorders. Particularly in the context of questions put forward by this study in conjunction with other psychological influences (of social networks and of social support), it is advisable to question how the specific behaviour or patterns of daily interaction drawn from EE are featured in the strength of appearance of the different disorders. Similarly it should be questioned how the corresponding programmes of intervention are thus attuned.

There is a number of prospective studies that confirm the link between EE and the development of and recovery from an illness, as well as the high value of EE in schizophrenic prognoses.

In an overview of 27 studies that examined the relapse rate of patients influenced by the EE status of their families, Kavanagh (1992) points to an average relapse rate over all results of 21% for low EE surroundings and 48% for high EE families. This effect does not come back to possible confounding influences such as how chronic the disorder is, but is however dependant on the application and restrictions of the concept. In this way, these studies are at their best when using a complex interactive model of relapse i.e. interactions between symptom-induced behaviour, other stress factors and Expressed Emotion. Another factor that contributes to an increased relapse rate is the frequency of contact with high EE relatives. Although not all the studies' findings are confirmatory (several summary studies are replications), the newer results are above all consistent.

the authors to conclude that a lack of potential support could have just as negative an effect as tense and stressful relationships.

In a meta-analysis from 25 worldwide studies (N=1346), Bebbington and Kuipers (1994) were able to confirm the strong effect of EE as a predictor for relapses in schizophrenic disorders. The global relapse rate here stands at 50% for high EE families and 21% for low EE surroundings. In total, the proportion of cases with EE values stands at 55%, in accordance with the customary cut-off. When comparing EE, medication and relapse rates, results show that EE has a more potent effect than medical therapy, whereby the extent of the link between EE and relapse rates is comparable in groups with medication (high EE 44.3% / low EE 18.4%) and groups without the aid of medication (57.7% / 27.9%). Similarly, the general prospective role of EE (being able to determine relapse frequency) is seen equally in men and women. Moreover the association of EE with relapses more evident in families that show a higher contact frequency with the patients (58.8% / 41.7%), whereas families with low levels of EE contact (18.3% / 23.9%) seem to have a prophylactic effect.

The proven connection between informal emotional environments in cases of schizophrenic disorders and other illnesses was comparatively under-researched, after original confirmations of two relatively early studies (Vaughn & Leff, 1976; Leff & Vaughn, 1980) to do with depression.

The only studies that actually made a direct comparison of relapse rates of schizophrenic and depressed patients exposed to either high or low levels of EE are those carried out by Vaughn & Leff (1976) and Leff & Vaughn (1980). In 1976 this happened especially with the viewpoint to also examine the influence of EE on the probability of relapses of depressed persons, which had previously only been proven for schizophrenic psychoses. The later study was designed to make a comparison of the two disorders.

The study of 30 unipolar depressed patients and 37 schizophrenics by Vaughn & Leff (1976) showed that 67% of depressed patients who lived with families with high levels of EE suffered relapses within nine months, whereas only 22% fell ill again who lived with low levels of EE. Similar results were found for the group of schizophrenics, in that 50% of patients in a high EE environment suffered relapses, and only 12% of patients in low EE surroundings. For the depressed patients a particular result was noticeable in that it was exclusively the EE dimension "criticism" which marked the high EE families, whereas for schizophrenics both "criticism" and "emotional over-involvement" were involved. The authors traced this state of affairs back to the fact that over-involvement is particularly indicative of parental relationships, and depressed persons live more often with partners than with their parents, as compared to schizophrenics. Leff & Vaughn (1980) replicated and broadened these findings in that they highlighted critical life experiences from the same 30 depressed and 37 schizophrenic people, in addition to EE in the families. It found that the relapses of schizophrenic patients in high EE surroundings were not preceded by any critical life experiences, whereas those of patients in low EE surroundings were. Relapses of schizophrenics are thus due to either high levels of EE or the occurrence of critical life experiences. Results for depressed patients turn this pattern on its head. For most patients in this group, it is the occurrence of a critical life experience together with a high EE environment that was linked with a renewed phase of depression. Consequently a vulnerability model can be affirmed for depressed persons, while there seems to be a direct influence of EE or external stress factors on the occurrence of illness for schizophrenic patients.

In a study by Hooley et al. (1986) similar results were shown in the relapse rates of depressed persons in high EE environments. Hooley et al. examined 30 clinically depressed patients and their partners. Altogether 59% of patients with partners with high

EE values suffered relapses, whereas no cases of relapses were found in partnerships with low EE values. Above all, the negative, critical behaviour patterns of partners are similar to the results of Vaughn & Leff (1976), which encouraged relapses in high EE conditions. As has already been confirmed above, a positive link can be determined between criticisms or over-involvement ascertained in interviews (CFI) and how the illness develops i.e. the risk of relapse in cases of depressive disorders.

Originating from the criticism that EE reflects above all the attitudes of relatives towards the patients, it was pointed out that, particularly within the scope of depressive disorders, the actual family interactions and in particular the interactions with the partner represent significant factors in assessing levels of EE. In two studies by Hooley & Teasdale (1989) and Hooley and Hahlweg (1989) similar results were found in that marital stress and frequent arguments are associated with high EE values and contribute independently to relapse rates. Moreover, on the one hand, conversations by couples with high levels of EE often descend into a vicious circle and on the other hand, the patients participate actively in this process through their interactive behaviour. In a study that observed groups of patients and relatives in connection with EE, Florin et al. (1992) discovered that there is a high level of reciprocity between patients and their partners with regards to EE and that in relationships with high levels of EE the patients, as well as the partners, show a high measure of negative behaviour. An increased correlation between the degree of depression and the EE values of the patients and their partners can also be shown. As a result, depression, or the appearance of depressive symptoms, has to be considered in itself a possible confounding factor. This is in the sense that more depressed persons cause or set off EE-related behaviour in each of their relatives as compared to less depressed or normal persons. Alternatively, that relatives' behaviour typical of high levels of EE depends on other forms of the manifestation of depressive symptoms. The

prevalence of a partner's EE status for depressed patients stands with 65% just as high as the values mentioned above.

In a large-scale longitudinal study, Fiedler et al. (1994) emphasise particularly the two-way character of the Expressed Emotion construct in depressed patient groups. In a pairing of EE analyses of patients and partners with an interactions analysis, highly negative patterns of interaction and communication were found in couples with high levels of EE. However in this case these seem to be independent of the manifestation of depressive disorders.

Altogether it is noticeable that in social environments which are marked by a high degree of EE and are shown to have stress- and relapse-inducing effects, an interactive framework exists particularly for depression, in which numerous patient variables have influence. In order to intervene effectively, it would be necessary to observe the existing patterns of interactions between patients and relatives, along with the relatives' behaviour, and to positively influence them where needed.

2.6.12 Psycho-social intervention based around the EE concept

An area where research into Expressed Emotion shows most effect is that of intervention. Here measures and concepts of EE serve to structure and evaluate psycho-social forms of treatment with families.⁷

There are now a number of successfully monitored studies of intervention with a two year follow-up (e.g. Leff et al., 1989, 1990a; Falloon et al., 1982, 1986; Tarrier et al., 1988,

⁷ An overview of intervention studies can be found in Lam (1991) and Kavanagh (1992). Detailed manuals of this form of family intervention, which link mostly EE-specific forms

1989.) In these studies, the relapse rates of the intervention group were no higher than those that could be expected from patients with low EE families, whereupon success could not be solely traced back to an improved family atmosphere, but also to an increase in the patients social competencies (Hogarty et al., 1986) and the reduction of intra-subjective strains (Falloon & Pederson, 1985). However, effective interventions were only sometimes linked to a direct reduction in EE or negatively affecting behaviour (Leff et al., 1982, 1990a; Hogarty et al., 1986). Furthermore, less successful studies could not reduce the level of EE in relatives, which leads to the conclusion that reducing Expressed Emotion represents an evaluative, but not a necessary part of recovery or reduction in the relapse risk.

There have however been a number of common factors or goals, over and above various programmes that make a difference to the success of intervention:

- Positive attitude towards the relatives. Do not incriminate the family or blame them.
- Clarification: offering a comprehensive set of instructions is a prerequisite of future intervention.
- Problem-solving: offers of problem-solving and communication strategies, which are relevant to current problems in the family.
- Emotional ways of coping: sometimes also called cognitive-behavioural strategies, which should allow a way of dealing with different situations of stress, strain and anger.
- Medication: All programmes try to help patients to maintain an optimal level of medication while they receive psycho-social treatment.

of intervention with parochial psychiatric practices and structures include Falloon (1985), Anderson et al. (1986), Barrowclough & Tarrier (1992) and Atkinson & Coia (1995).

However, the original hope that a purely psycho-educative form of intervention would be sufficient to positively influence the development of psychiatric disorders in the long-term could not be fulfilled. The corresponding findings show that clarification is an important start to subsequent interventions and represents a high value for relatives, but cannot influence long-term results as a lone measure (Tarrier et al., 1988; Lam, 1991). Changes with regards to a single aspect can be helpful, but the most successful programmes are those that modify the behaviour of both the relatives and the patients in interactions. In this way studies that only highlight one viewpoint, like social behaviour or negative symptoms, do not necessarily also improve the family environment (Tomaros et al., 1988) and vice-versa.

A small number of studies have investigated the relationship between expressed emotion and outcome of bipolar disorder, particularly relapse. Low levels of EE in relatives have been associated with lower rates of hospitalisation when compared with patients with relatives with high EE (Honig et al., 1997). However we should be very careful in interpreting findings based on rehospitalisation as admission to hospital may be confounded by EE and may not be a true indication of relapse. For example, relatives with high levels of EE may be less tolerant of symptoms and less willing to manage the patient at home than low EE relations (Hooley, 2007). Nevertheless, low levels of EE have also been shown to be a predictor of better outcome and higher overall functioning (O'Connell et al., 1991). Priebe et al (1989) found that individuals with bipolar and schizoaffective disorder living with a high EE relative and taking lithium demonstrated a poorer treatment response during a 3 year pre-interview period and that this trend continued even more strongly in the 9 months following interview. In a study of 23 hospitalised patients with bipolar or schizoaffective mania, levels of negative EE within

families approached significance in the prediction of relapse (Miklowitz et al., 1988). Family affective style was significantly predictive of relapse. A negative interactional style (critical, intrusive and guilt-inducing statements) by relatives was significant in predicting relapse. For those relatives scoring negatively on both measures, relapse rates were 94%.

As an extension to their studies of IFIT (Miklowitz et al., 2003b) and of FFT in the Colorado treatment/outcome study (Miklowitz et al., 2003a), Kim & Miklowitz (2004) considered the role that EE plays in moderating the success of a family-based psychosocial intervention. They broke the concept of EE down into two types of emotional distress: level of critical comments; and emotional involvement. They identified patients undergoing family therapy as either high or low EE. There was no difference between the two groups on relapse rates, nor was there any significant interaction between the type of treatment and family EE status. During follow-up a relationship was found between high EE families and high levels of depression unrelated to treatment group; in particular there was a main effect of critical comments on depressive symptoms. These findings were not replicated with manic symptoms.

Simoneau et al. (1998) investigated family communication in individuals receiving family-focussed psychoeducation and crisis management. They found that treatment condition and levels of EE contributed independently in positive family communications at 1-year follow-up. However, treatment did not have a significant impact on EE in family members, or on the verbal behaviours of participants. However, our consideration of interactional behaviours should perhaps be widened to include non-verbal behaviours, which were found to moderate symptom improvement. Non-verbal behaviours improved significantly following family therapy.

Negative results have also been found in relation to EE and outcome, with Koenig et al. (1997) finding a relationship between self-rated critical or intrusive relative's statements and distress, but not with symptoms. Miklowitz et al. (2005) found that the severity of relatives' critical comments did not predict symptomatology, but that distress caused by criticism was associated with increased symptomatology and days unwell. The effects of EE may also be mediated by other factors including personality traits, self-esteem and dysfunctional attitudes (Yan et al., 2004). This may explain why the relationship between expressed emotion and symptomatology is not always clearly defined.

A differential relationship of EE to depressive and manic symptoms, as found in Kim & Miklowitz's study, was replicated in a 1-year longitudinal study of 47 out-patients with bipolar I disorder (Yan et al., 2004). EE, perceived criticism and negativity of a significant other was measured. High EE relatives predicted depressive relapse but not manic, this finding was maintained when hypomanic episodes were included and prior symptom severity was controlled for. Patients experiencing high EE were 5 times more likely to face depressive relapse. These findings add support to a polarity specific relationship with EE, which has implications for intervention.

Impact upon EE can only be achieved through a holistic approach involving individuals whose behaviour directly touches the patient. Family therapy developed from this premise. As discussed above, family therapy has been found to be effective in improving symptomatic outcome (Miklowitz et al., 2000), increasing time to relapse (Miklowitz & Goldstein, 1990; Miklowitz et al., 2000; Rea et al., 2003), preventing hospitalisation (Rea et al., 2003), and improving medication compliance and functioning (Clarkin et al., 1998). In a trial of family therapy for bipolar disorder, Honig et al. (1997) assessed levels of expressed emotion. Multi-family psychoeducation was provided for 29 families over 6 bi-

weekly sessions. Treatment focused on the provision of illness related information, methods of coping more effectively with illness, and recognition of the need for support for both patients and family. Though not directly focusing on EE it produced a significant reduction in the levels of EE in participating families compared to a waiting list control receiving no intervention. A psychoeducational intervention for relatives consisting of two four hour sessions, following group therapy for the patients, was followed by significant reductions of EE at 1 year follow-up (Bernhard et al., 2006). However, a causal link cannot be ascertained as it may be that changes in the patients' behaviour lead to this reduction.

The mechanisms by which expressed emotion affects the individual with bipolar disorder are still relatively unknown, as are the ways in which it develops in relatives of patients. It has been suggested from studies examining the interchanges between the bipolar patient and relatives, that there is 'reciprocal negativity' in the interactional patterns of the family of these patients (Simoneau et al., 1998). Similarly, Rosenfarb et al. (2001) in a 9-month prospective follow-up found that patients' unusual thinking and relatives' harsh criticism was more likely to be correlated when patients relapsed, so that residual symptoms had an effect on the interactional style of the relative. Miklowitz, Goldstein & Nuechterlein (1995) found an association between negative affective style of relatives, based on observer ratings, and an oppositional style in outpatients at a hospital. The authors concluded this may reflect residual symptoms rather than a habitual style of interacting. It may be then, that relatives are reacting at least in part to residual symptoms of the patient. Wendel et al. (2000) found that relatives with high expressed emotion were more likely to believe the patient has control over his/her symptoms. They also tend to have a more internal locus of control for their own behaviour than do low-EE relatives (Hooley, 1998). Wendel et al. (2000) hypothesise that this attribution leads to more

criticism, in attempt to bring the behaviour under control, thus leading to increased stress and contributing to symptomatic deterioration. A study of a group with mixed diagnoses lends more support to the attribution hypothesis – i.e. relatives having higher expectations of their family member leads to high EE. Heikkila et al. (2006) interviewed 42 first-episode patients with schizophrenia-related psychoses and mood disorder with the five-minute speech sample method (Gottchalk & Glaser, 1969). Stepwise regression analyses were used to analyse the relationship between EE, sociodemography, cognitive performance and psychopathology. Premorbid childhood adjustment and cognitive variables were the only variables significantly associated with EE.

Hooley & Gotlib (2000) propose a diathesis-stress model of EE to explain both the development of EE in relatives and the impact of high EE on the course of patients' disorders. Their paper, which focuses on clinical outcome in schizophrenia, depression, and borderline personality disorder, recognises EE as a relational variable i.e. a product of the interaction of both patient and relative characteristics.

Criticism has been aimed at the methodology of expressed emotion studies, including the small samples and heterogeneity of diagnoses and treatment methods. Research on expressed emotion has also been criticised for using criteria inferior to the DSM-IV and not distinguishing between depressive and manic relapse (Yan et al., 2004). In addition, most research on EE in bipolar disorder has assessed the presence or absence of EE on the basis of observer ratings of family interactions. Koenig, Sachs-Ericsson & Miklowitz (1997) found that while observer ratings of harsh and benign criticisms correlated with patient ratings, there was a difference in the way in which observers and patients viewed intrusive and supportive statements. It could therefore be fruitful to include assessment of the patient's perception of the interaction.

Further caution should be exercised in drawing conclusions about the causal role EE plays in relapse. Although it is plausible that high levels of EE may cause vulnerable patients to relapse, it is also conceivable that some of the characteristics of relapse-prone patients might provoke criticism in family members (Hooley, 2007). Thus the direction of causality should not be assumed, if indeed a causal relationship exists at all. However studies in schizophrenia do not support this assumption. The literature suggests that EE is not just a reaction to specific characteristics of patients. The fact that different relatives within the same family can have different levels of EE further supports this (Weissman et al., 2000).

In future research there may be merit in examining the types of critical remarks made in order to aim at specific targets for intervention. This could explain the polarity specific effect of EE. In a study by O'Brian et al. (2006) on adolescents at risk of developing psychosis, it was found that the majority of critical remarks were made about negative symptoms and irritability/aggression. It may be that in bipolar disorder the majority of critical remarks are focused on depressive elements of the illness. This could prove important in the development of specific therapies targeting the nature of critical remarks.

Finally, it is interesting to note that whilst the relatives of patients with schizophrenia tend to be parents, in mood disorders it is more likely to be spouses that patients return home to live with. Emotional overinvolvement, a key element of EE, is more commonly noted in parents than it is in spouses (Goldstein et al., 2002). This offers a potential explanation as to why EE is more pronounced in families of patients with schizophrenia, although the small number of studies in bipolar disorders makes conclusions difficult to derive.

2.6.13 Social Support and Expressed Emotion

In both of the concepts discussed above, i.e. Expressed Emotion and social support, it is about trying to encapsulate social living environments and, to a limited extent, psychiatric and parochial psychological discourses and to make more accessible. On both sides, social factors with different focal points are implicated in the development of psychological disorders, whereby the common ground shared by both projects consists in the fact that both approaches do not deem individual-centric factors to be central in the formation and development of psychological disorders. Instead, they basically observe the individual's interaction and social environment, emphasising here the significant influence of psycho-social stress factors. Further commonalities exist between the concept of "critical life experiences" and other significant socio-economical factors.

In the context of Expressed Emotion mainly negative and burdensome aspects of social relationships are highlighted. These may consist of rejection, excessive demands and a lack of distance, but not the healing effects of social attachments. In previous research, the potential effect of social support or the negative influence of high levels of EE were observed mostly only when clearly separated. These two aspects were only seldom analysed in their interdependence and mutual overlapping. In recent years, however, research into social support has paid more attention to the negative and burdensome aspects of interactions. For instance, a search for literature in the psycLIT database carrying the keywords Expressed Emotion and social support found only 8 studies between 1974 and 1996. In fact only two of these studies really deal with both concepts and then only one compares them with regards to their influence on psychological health (Franks et al, 1992; Hahn et al., 1995). These findings become more understandable when

the different research traditions of both concepts are taken into consideration. The EE concept is normally applied in a context of family therapy with regards to a specific and serious disorder, schizophrenia. Conversely, the concept of social support has its roots in primarily epidemiological studies on critical life experiences and social networks with "normal" people. Whereas EE is normally used with regards to forms of development or relapses in clearly defined disorders or clinical conditions, social support is associated more with other aetiological or epidemiological problems.

Aside from the findings of EE research, results on the burdensome effect of negative aspects of social support advocate that social strains in the sense of negative interactions make an independent contribution to the understanding of psychological disorders. In some cases, it is even in the position to account for more variance than factors of positive social support alone (Firoe, 1983; Rook, 1990, 1992; Sommer & Fydrich, 1989; Coyne & Bolger, 1990). It is also presumed in research, which concerns the connection between family structures and psychological health that it is more the presence of negative interactions, which separates the durable relationships from the dysfunctional, than the absence of positive or negative interactions. Moreover, aversive and negative relationships have a more forceful influence on psychological well-being than supportive ones, and that the absence of negative support explains a lot about the link between social support and psychological health (Franks et al., 1992). As an example, in a large, epidemiological study of depression, single or divorced people, most of whom do not have a close confidant, have double as high values of depression as those who are happily married. However those in an unhappy partnership reached an approximately 25 fold increase in their levels of depression (Weismann, 1987).

Furthermore interesting results are to be found when observing the social surroundings of psychiatric patients. For example, Sarason & Sarason (1982) considered the link between perceived social support and general attitudes to psychological disorders. Relatives questioned and primary contact persons of psychiatric patients, who themselves show a high perceived level of social support, seemed more congenial to people with psychological disorders compared to relatives with low levels of perceived social support and were generally critical of long periods of hospitalisation. Similarly, differences were found in the stances taken on psychologically ill people, connected with EE. Thus relatives with high levels of EE are generally of the opinion that the patient has considerable control over his or her behaviour and is responsible for their illness. Those relatives with low levels of EE however show a higher level of understanding of patients, and attribute undesirable behaviour as well as the illness itself more to external factors (Leff & Vaughn, 1981).

Franks et al. (1992) examined 83 patients from a doctor's surgery that had all been treated for heart-circulation problems. They measured in essence the perceived EE, perceived social support, negative life experiences in the last three months and gauges of psychological health, above all depression. The values of depression were correlated positively with perceived criticism, EE, ($r=.38$). The result was an inverse correlation with the measures of perceived social support ($r=.39$). Additional regression analyses showed that critical life experiences and social support clarify a substantial part of the variance of depression measures ($R^2=29\%$), that once under the umbrella of the EE variables, the share of explained variance increased ($R^2=38\%$) and that the variables of social support were no longer significant i.e. played no more role in clarifying variance. To the exclusion of life experiences gathered, the same pattern emerges i.e. the role of social support did not become significant and the share of explained variance of EE measurements dropped

to $R^2=18\%$. The style of interaction recorded in families with EE gauges were described by Franks and colleagues as negative social support and conclude, in accordance with their regression analyses, that the absence of this negative social support is more significant than the presence of more positive social support. The specific quality of the relationships in the family, primarily whether they are critical or over-involved, has a stronger influence on psychological health than the social support received does.

The second study by Hahn et al. (1995) that considered the connection between EE and social support had a fundamentally different design. 20 alcoholics and their partners were studied, as well as 21 control couples. For all participants, the EE indices were obtained using the Five Minute Speech Sample, perceived social support and psychological symptoms with a symptom checklist. The EE status of partners of alcohol-dependant patients differed significantly from partners in the control group, and a clear correlation between the EE status of the alcoholics and their partners ($r=.38$) emerged. Similarly, the alcohol-dependant patients report a lower level of social support and a significantly higher level of partnership strain than the control persons. At the same time, in the context of EE and social support, a substantially negative correlation between the EE status and generally perceived social support for the group of alcoholics on the one hand ($r=-.59$) and on the other a significantly positive correlation with the measure of social strain ($r=.50$). Patients with high levels of EE felt correspondingly less supported and more under strain than patients with a low EE index. The same goes for the EE status of partners. Patients with partners who have high levels of EE feel correspondingly less supported and more under strain from the respective partner. Similarly comparable correlations were also to be found in the control group, in that male control persons with high levels of EE report a heavy social strain from their partner ($r=.62$). In the same way, women with high EE indices demonstrated a negative connection to perceived social

support ($r=-.60$) and a significant positive correlation with subjective partnership strain ($r=.48$). However, no links to measures of psychological health were reported. It can still be said that there is a significant mutual dependence or overlapping of perceived social support or partnership strain and EE status, even if nothing about the type of links within this design can be shown i.e. whether the perception of social support is influenced by behaviour linked with high levels of EE or vice-versa.

The discussed examples suggest that both concepts of perceived social support and received Expressed Emotion represent, in many areas, self-enhancing and self-overlapping features of social attachments and living environments that cannot be isolated from another. The specific type of support, source of support, characteristics of the support and the respective personal situation are vital in deciding how supportive or burdensome social relationships are experienced or come across.

Regarding psychological health of groups with increased levels of vulnerability, the constancy of remissions, the seriousness of symptoms and the probability of relapses are heavily influenced by positive and supportive aspects of social interactions, as well as negative and burdensome ones. In the context of research in the field of EE, results showed that social relationships can be so burdensome that they have a negative effect on the course of the illness or psychological well-being. It can be assumed that in this way a negative family atmosphere also has an influence on the scale of the social support offered and perceived. Moreover patients will judge actual help more negatively or experience subjectively less emotional support. Conversely, high EE relatives will also regard the patient as more of a strain, which in turn affects their reactions towards the patients and their social network. At the same time families with low levels of EE are characterised rather in that the patients are proven to have a lower rate of relapses and

longer remission phases. However up to now it has not been shown which behaviour traits distinguish low EE relatives and have healing effects. This is because it has been shown that low EE relatives who at the same time display an emotional indifference towards the patients greatly increase the vulnerability of the patients to stress factors outside the family unit (e.g. Leff & Vaughn, 1980). Presumably results here show similarly important conceptual overlaps between helpful dimensions of social support or describable supportive behaviour patterns.

In any case, the magnitude of Expressed Emotion seems to present a useful possibility to understand negative or burdensome aspects of attachments and structures of interaction, in connection with the concept of social support alongside the healing capabilities of social networks. Consequently it is conceivable that both these aspects of social environments i.e. negative and positive qualities, are related to the same variables influencing the psychological health of high risk patients, such as psychiatric patients. In order to find out more about the respective processes of disorder formation, course and recovery, it would also be a good idea to consider these two aspects in cases where EE or features of social support are implemented in interventions or preventative programmes with patients groups of increased vulnerability and high risk of relapse.

2.7 Psychological Factors

2.7.1 Dysfunctional Attitudes

As with other psychosocial and psychological factors, it is difficult to establish the direction of causality with cognitive variables and bipolar illness. Whilst it is possible that

cognitive differences are a consequence of prolonged illness, it is also likely that they are part of the cause of illness onset

Research has progressed into establishing a pattern of cognitive deficits in individuals with bipolar disorder. The presence of dysfunctional attitudes in affective disorders has been investigated in accordance with Beck's cognitive therapy (1976) and cognitive model of affective disorders. One set of studies has focused on dysfunctional attitudes in euthymic individuals. Scott and Colleagues (2000) simultaneously examined a range of cognitive factors taken from Beck's model of cognitive vulnerability (1976). They compared 41 euthymic bipolar patients with 20 healthy controls. Despite the patients being euthymic they showed significantly higher levels of dysfunctional attitudes, as measured by the Dysfunctional Attitudes Scale (DAS, Weissman & Myers, 1978); particularly on the perfectionism and approval sub-scales. The significant finding was independent of IQ and depression level. Previous studies have also found this increased perfectionism and have associated it with poor acceptance of the disorder and poor adherence to medication (Scott, 1995).

As cognitive abnormalities are present in euthymic periods it implies that patients do not achieve full functional recovery even when symptom free. Support for Scott's findings came from Lam and colleagues (2004) who also found that dysfunctional attitudes are still prevalent in euthymic bipolar patients. They identified a subscale within the DAS which targeted goal-attainment behaviours, when an individual has unrealistic beliefs about having to be happy all the time and having to excel at all things without excessive effort. They suggest dysfunctional attitudes are a risk factor for a bipolar episode as this subscale was positively correlated with past number of hospitalisations and number of previous bipolar episodes. Rosenfarb et al. (1998) found that women with bipolar who were either

depressed or remitted were more self-critical than controls. The authors suggest these findings are evidence of self-criticism as a relatively stable personality trait. These studies do not rule out the possibility that dysfunctional attitudes emerged as a result of the disorder. Hollon, Kendall & Lumry (1986) found that remitted bipolar patients had no evidence of elevated depressogenic processes. These different findings may reflect differences in definition of the beginning of euthymic periods.

Scott & Pope (2003) examined differences in cognitive patterns in different phases of bipolar disorder. They investigated dysfunctional attitudes, self-esteem and personality style in remitted, depressed and hypomanic patients. They found euthymic patients had the lowest score for dysfunctional attitudes and sociotropy. Depressed patients had the highest number of dysfunctional attitudes and the hypomanic patients fell in between these two groups. The study however was limited by its small sample size. Following research that suggests that dysfunctional attitudes change in accessibility depending on mood, Wright et al. (2005) deliberately improved or worsened mood in 120 individuals with remitted bipolar I disorder, remitted unipolar depression, and a control group. The results showed that increasing mood reduced dysfunctional attitudes less in the bipolar group compared to the other two groups. These results fail to support the mood-dependency theory, which suggests that high mood activates manic schema. The suggestion here is that despite high mood, bipolar individuals have an inability to relax unhelpful assumptions which may then interact with elevated mood to further increase mood levels. This failure to return mood to baseline levels appears to fit with the behavioural activation system theory.

The next set of studies examined cognitions specific to the manic phase. Lam, Wright & Sham (2005) developed The Sense of Hyper-Positive Self Scale (SHPSS), which measures

the extent to which bipolar patients value themselves and perceive themselves to possess attributes such as persuasiveness, productiveness and dynamism. In all 51 patients received Cognitive Therapy and 52 entered the control group who received mood stabilisers plus regular psychiatric outpatient care. In regression analysis which included the Mania rating scale, Beck Depression Inventory and the DAS goal attainment subscale as independent variables, Sham (2005) found that the DAS goal attainment subscale contributed significantly to SHPSS scores. Individuals receiving CT were significantly less likely to experience bipolar episodes and spent significantly fewer days in bipolar episodes than the control group. Patients with high SHPSS scores were significantly more likely to relapse, and that also such patients found cognitive therapy to be less effective. Thus, goal-attainment appears to be linked to attributes found in individuals experiencing a manic episode. Goldberg et al. (2005) also examined dysfunctional attitudes in mania. They compared cognitions of 23 bipolar manic or hypomanic patients, 28 patients with unipolar depression and 24 normal controls using the Cognition Checklist for Mania (CCL-M) which measures maladaptive cognitions and beliefs associated with mania. Unsurprisingly the total CCL-M score was significantly higher for the bipolar manic than the unipolar depressed patients. However, there were differences in the subscale scores of the bipolar and unipolar patients with bipolar patients endorsing cognitions and beliefs about excitement and past and future memories significantly more than unipolar patients.

Two studies have investigated attributions and underlying information processing in the manic phase. Thompson and Bentall (1990) found a relationship between hypomania in a healthy population and the tendency to make excessively global attributions to both positive and negative events. The authors hypothesise that if hypomania were a defensive stance against negative events, one would expect a pattern similar to depression, i.e. excessively global, stable and internal attributions in relation to negative events, which

was not found. Lyon, Startup & Bentall (1999) found that manic patients showed a self-serving bias and endorsed more positive than negative traits for self, the same as healthy individuals. However, they also showed slow colour-naming for depression related and not euphoric words, and recalled mainly negative words. The authors take this as being broadly consistent with Neal's (1998) account of the manic defence, where individuals become defensive in response to negative stimuli.

Jones et al. (2005) compared dysfunctional attitudes in individuals with bipolar I disorder, unipolar and controls. Those with major recurrent depression showed the highest level of dysfunctional attitudes, followed by bipolar disorder, with the least being shown by controls.

However not all studies show a relationship between dysfunctional thinking and bipolar illness. Using a prospective study design, Tzemou & Birchwood (2006) found that when mood symptoms and phase of illness were controlled for, bipolar patients were largely indistinguishable from controls on measures of dysfunctional thinking.

It has been proposed that negative cognitions predict the course of bipolar depression. One longitudinal study found that cognitive style (including dysfunctional attitudes) interacts with stressful life events to predict depressive and manic symptomatology in unipolar and bipolar undergraduates with no prior treatment history (Reilly-Hamilton et al., 1999). However, other prospective studies looking at dysfunctional attitudes and automatic thoughts and their relationship to bipolar symptoms found that cognitive scales were related to current and predicted depression, but not mania (Johnson & Fingerhut, 2004). Safford et al. (2007) suggest that underlying negative cognitive style may account for the stress generation effect often found in depressed individuals (that depressed

patients generate more negative life events). They propose that addressing cognitive patterns in treatment may not only reduce depression, but following the stress generation theory, may also reduce the likelihood of experiencing negative events.

A review of psychosocial predictors of bipolar disorder by Alloy and colleagues (2005) concluded that there is consistent evidence that cognitive style predicts the onset and recurrence of bipolar episodes, and that this seems to be mediated by life events. One methodological point is that people may experience different accessibilities of cognitions at different times e.g. negative cognitions difficult to access in periods of remission. There will also likely be problems with distractibility in periods of mania (Lyon, Startup & Bentall, 1999).

2.7.2 Self esteem / Self concept

Evidence for the importance of self-esteem in bipolar disorder comes from studies looking at levels of self-esteem at various phases of the disorder, and those exploring a relationship between self-esteem and relapse. Significant differences have been found in measures of self-esteem between depressed individuals and remitted or healthy individuals. Hypomanic individuals show a level of self-esteem in between the two which consists of combined positive and negative self-esteem (Scott & Pope, 2003). Shapira et al. (1999) found that individuals in remission from bipolar disorder for 12 months reported poorer self-esteem than matched controls. Blairy et al. (2004) found in a sample of 144 patients with bipolar disorder, that their self-esteem was significantly less than controls after at least three months in remission. These findings have been supported by other studies (Serretti et al, 1999; Himmighoffen et al., 2003; Serretti et al, 2005). However other studies have found no significant differences between the self-esteem

scores of bipolar patients and normal controls, (Bauwens et al, 1991, Pardoen et al, 1993; Scott, 2000, Daskalopolou et al., 2002).

One study examined the different components of self esteem in affective disorder compared to those without affective disorder. Serretti et al. (2005) examined levels of self esteem in bipolar disorder, major depressive disorder and controls. Self esteem of euthymic individuals with affective disorder was significantly poorer than controls. However, following the factor analysis, the self-esteem profile of bipolar and upipolar individuals was found to be identical, with both self confidence and self deprecation being significantly lower than controls. In a subgroup with low self-esteem, bipolar and unipolar patients had a lower level of self-confidence but a similar degree of self-deprecation. The authors suggest that those with affective disorder lack the self attribution of positive characteristics. These findings were supported by Jones et al. (2005) who found that both bipolar and unipolar individuals had poorer self esteem than controls, but were not significantly different from each other, when current levels of depression were accounted for.

One difficulty in defining the relationship between self-esteem and bipolar disorder, is the number of overlapping concepts related to self-esteem. Pardoen and colleagues (1993) found a tendency to socially conform in individuals with bipolar disorder and suggested social adjustment as a mediating factor. This hypothesis is supported by Staner et al. (1997) who found that the relationship between self esteem and new bipolar episodes was confounded by the effect of social maladjustment (Staner et al., 1997). This relationship has also previously been found in unipolar patients (Pardoen et al., 1993). However, Shapira et al, 1999 found that although adjustment difficulties of unipolar patients were associated with poor self-esteem, social adjustment was impaired in bipolar

individuals with no self-esteem deficit. Winters & Neale (1985) suggest that individuals with bipolar disorder do have negative feelings about the self, but that explicit measures of self-esteem don't elucidate these feelings. However when more implicit measures of social desirability and self deception are carried out they score much higher on measures of low self-worth. Scott & Pope (2003) also support the view of the 'manic defence', where threats to self-esteem are kept away by grandiose thoughts, and result in high scores on explicit measures of self-esteem. In support of this view, Bentall & Thompson (1990) found that individuals with hypomanic traits took longer to name depressive related words than people without hypomanic traits. They also were more likely to have higher scores on the Beck Depression Inventory. It appears that self-esteem may be interrelated with a number of concepts. Hayward et al. (2002), in an exploratory study, found that individuals with bipolar disorder and low self-esteem were also more likely to feel stigmatised by their illness. They suggest a dual relationship where low self esteem increases feelings of stigmatisation, and vice versa.

As levels of negative self-esteem increase it is thought to be a risk factor for relapse into bipolar depression (Scott & Pope, 2003). It is also associated with a greater risk of suicide (Daskalopoulou et al., 2002). It is unclear whether low self esteem is caused by depression or whether low self-esteem causes an underlying vulnerability to depression. However, since low self-esteem has been found in remitted individuals, it would suggest a more stable characteristic (Blairy et al., 2004). Longitudinal studies would be required to show a causal relationship.

Information processing may have a part to play in maintaining high or low self-esteem. Murphy et al. (1999) found that manic patients show an affective bias for positive stimuli, whereas depressed patients show an affective bias for negative stimuli.

Other factors may affect self-esteem, for example alcohol, particularly in females (Salloum et al., 2001). Self-esteem was found to be a mediating factor in the relationship between low social support and changes in depression in those with bipolar disorder (S.L. Johnson et al., 2000).

In order to interpret studies of self-esteem correctly it is important that they take account of current levels of symptomatology (Scott et al., 2005). Even when participants are judged to be clinically euthymic, results may be confounded by sub-syndromal symptoms.

In summary, the evidence highlights the importance of self-esteem management in the long-term course of bipolar disorder.

2.7.3 Affect and emotion related personality traits

Research has examined the differences in the bipolar personality structure from normal controls. Roy (1990) found that depressed patients, including unipolar patients, scored higher for introversion, neuroticism, hostility, and significantly lower on measures of self-esteem. In contrast, other studies have found there to be no differences in personality traits from healthy controls (Clayton et al, 1994). Most research has studied bipolar individuals in different stages of their illness, making it difficult to establish causality or to make a comparison with non-affected individuals.

Comparisons have been made between the personality of individuals with unipolar and bipolar illness. Bech et al. (1980) found that there were more similarities than differences

between the personality patterns of unipolar and bipolar patients. This study was unusual in drawing both the unipolar and bipolar samples from 'manic-melancholic patients'. Bech and colleagues point to the high number of lithium responders within the unipolar sample as a possible explanation for the similarities between unipolar and bipolar individuals. Bagby et al. (1996) examined unipolar/bipolar differences in relation to the Five-Factor Model of Personality (Digman, 1990). The results showed that euthymic bipolar individuals scored significantly higher on the Openness (O) dimension and the positive emotions facet of the Extraversion (E) dimension than did euthymic unipolar individuals. The authors suggest that bipolar patients are therefore more likely to experience positive emotions and be sensitive to positive and negative emotions. Odegaard et al. (2005) compared type A behaviour pattern (characterised by time urgency, impatience, irritability and competitiveness) in a group of bipolar I and unipolar patients. They found that those individuals with bipolar I were more likely to be impatient, hurried, quick-tempered and easily irritated than unipolar individuals, and that this appeared to a stable trait.

A number of studies have compared personality traits of bipolar individuals in remission with a control group. Both Hirschfeld et al. (1986) and Solomon et al. (1996) selected four clusters of personality: emotional strength, interpersonal dependency, extroversion/introversion, and a miscellaneous scale, measuring energy, dominance, neediness, inflexibility and perfectionism. The first study, which also included a unipolar group, found that the bipolar group had less emotional strength (which included neuroticism) than the never ill control group. The authors found there was a gender split with regards to extraversion. Bipolar men showed normal levels of extraversion whereas bipolar women were more introverted. Bipolar women and men showed increased interpersonal dependency, compared to the never ill control group. Solomon et al

(1996), obtained similar results, finding bipolar individuals to be more “needy”. Mattusek & Feil (1986) assessed personality in remitted unipolar, bipolar and a non-depressive control group. The authors found that the bipolar group differed from the other two groups with regards to higher neuroticism than controls (although not as high as the unipolar group), hypomanic features, an obsessive-compulsive character, and a drive for success and achievement. Lastly, Scott et al (2000) using the Sociotropy-Autonomy Scale (Beck et al, 1983) found that euthymic bipolar individuals had higher levels of sociotropy (characterised by greater dependence on relationships with others) and autonomy (characterised by greater emphasis on independence) than healthy controls.

A group of studies has assessed personality vulnerabilities and risk factors in the course of the disorder. Research carried out by Lozano and Johnson (2001) looked at personality traits related to depressive and manic symptoms. They found that high neuroticism predicted depressive symptoms over time, and high conscientiousness in respect of achievement striving predicted manic symptoms over time. However, this small sample was not fully remitted and some characteristics may have been mood-related. Akiskal et al (1995) created a profile of individuals with major depression who would switch to bipolar disorder. They assessed the personalities of 559 patients, whom they followed up for a period of up to 11 years. Their newly created factors of Mood Lability, Energy-Activity and Daydreaming best described those who converted to bipolar II disorder. The authors suggest these are underlying traits, creating vulnerability to switching. Mood Lability may even “represent the very mechanism that underlies the ease with which switching into hypomania occurs.” Clinical characteristics also marked out Bipolar II switchers, such as high rates of substance abuse, educational, marital and occupational disruption, and minor antisocial acts prior to hypomanic episodes. In contrast, Bipolar I converters were distinguished only by greater acuteness, severity and

psychotic symptomatology. Other authors have studied the personality traits underlying the familial diathesis of bipolar disorder (Hantouche & Akiskal, 2005). It was found that mood dysregulation or “rapid shifts in mood and energy” held the biggest risk for a positive family history of bipolar.

In their review, Najt et al. (2007), identify impulsivity as being frequently associated with bipolar patients regardless of the phase of the illness. Impulsivity is described by Moeller et al. (2001) as a predisposition toward unplanned reactions to internal or external stimuli, without regard to the negative consequences. Among bipolar features, impulsivity tends to undermine mood stability, aggravate behavioural problems associated with mood lability, and can result in acting without forethought.

Further work needs to take place to elucidate the relationship between personality and bipolar illness. Personality measures may be picking up on subsyndromal symptoms, or on the other hand, may sample more permanent traits which act as risk factors for the illness itself. Given the research that has been mentioned on social rhythms, research could examine the relationship between personality and social rhythm instability. Meyer and Maier (2006) found that individuals with a hypomanic personality (as measured by the Hypomanic Personality Scale) and rigidity (a subscale of the Munich Personality Test) had a higher variability in sleep patterns. The study did not however go on to assess manic and depressive episodes, so could not assess how this variability in sleep affected aetiology. Other authors have linked personality characteristics such as extraversion and achievement striving with regulation of the Behavioural Activation System (Depue & Collins, 1999).

2.7.4 Impulsivity

Not strictly recognised as a personality trait, rather a 'complex behavioural construct' (Swann et al., 2005), impulsivity has been identified as a prominent and measurable characteristic of bipolar disorder. It has been observed to undermine mood stability in bipolar disorder and exacerbate behavioural problems (Henry et al., 2001; McElroy et al., 1996). It is associated with considerable morbidity, psychosocial impairment, accidents, suicide, and violence (Hollander et al., 2002). It seems to prevail during episodes of mania, but is also observed during periods of euthymia and other mood phases (Moeller et al., 2001; Swann et al., 2001, 2003). From their review of studies examining impulsivity in bipolar disorder, Najt et al. (2007) conclude that impulsivity has both state-dependent and trait-related features.

Swann et al. (2004) have shown impulsivity to be associated with substance abuse and suicide (Swann et al., 2005), two factors which have been previously shown to be associated with a poorer course of illness. Najt et al. (2007) conclude that further understanding of the role that impulsivity plays in the psychopathology of bipolar disorder could lead to improvements in the way we treat the illness.

2.7.5 Affect Regulation

Coping strategies may involve learning about self-regulation (of sleep, eating, exercise), adherence management and self-monitoring (of mood, thoughts and behaviours) (Scott, 2001). Research has examined the relationship between life stressors, coping and bipolar disorder. The process of coping is multi-faceted and may be linked with a number of psychosocial factors such as social support, parental style and prior experiences (Christensen & Kessing, 2005). Lam & Wong (2005) reviewed five randomized controlled

trials that included the early detection of prodromes and teaching of adaptive coping strategies. Four reported beneficial effects of delaying relapse, shortening episodes or hospitalisations. As discussed above, evidence suggests that interventions which target the identification of early warning signs and teaching patients coping skills to counteract them can be helpful in reducing the risk of relapse. Given that the prodromes can precede full-blown episodes by up to several weeks (Smith & Tarrier, 1992), such interventions can be beneficial in nipping an episode in the bud (Watkins, 2003).

There have been few studies on coping skills in the bipolar population, although many treatment trials have incorporated the teaching of coping skills, as previously discussed. In a naturalistic study in which 40 bipolar euthymic patients were interviewed, coping skills, along with current level of depression and ability to detect prodromes, were associated with level of social functioning (Lam & Wong, 1997). There have been other studies that indicate high levels of denial coping and low levels of acceptance coping are associated with poorer medication compliance and poorer outcomes in bipolar disorder (Greenhouse, Meyer and Johnson, 2000). These studies have been naturalistic in nature and as such causality cannot be deduced. In addition, coping strategies may be state-related (Christensen & Kessing, 2005) and therefore may be affected by subsyndromal symptoms.

A differential ability to detect prodromes has been detected, according to the type of relapse Lam & Wong (1997). A significantly higher ability to detect manic prodromes was reported than depressive prodromes. (7.5% could not detect manic prodromes versus a quarter for depressive prodromes). However, this difference finding was not replicated in a further study by Lam et al. (2001). However, the authors did find that patients reported difficulties in identifying depression prodromes, and that these prodromes were more

diverse and consisted of a mix of behavioural, cognitive and somatic symptoms, as opposed to manic prodromes which were mainly behavioural symptoms. Smith & Tarrier (1992) in a retrospective study found that individuals recruited from a self help group were slightly more able to identify depressive than manic prodromes. More depressive prodromes were also identified by participants of a prospective study (Keitner et al., 1996). The participants reported more insight in a depressive episode, as the 'good feelings' associated with mania may be misattributed to well-being, or accepted as a welcome respite from depressive symptoms.

It is important for prevention of relapse that individuals identify the content and meaning behind particular prodromes. Research suggests that individuals are able to identify idiosyncratic prodromes (Smith & Tarrier, 1992; Keitner et al., 1996). Type of prodrome may be different for depressive and manic relapses. One study found unusual thought content to be a reliable prodrome for mania, and conceptual disorganisation for depressive relapses (Altman et al., 1992). However, this study was based on a small sample of recently hospitalised patients. Lam, Wong & Sham (2001) reported that differential use of strategies at the prodromal stage predicted relapse when mood levels at baseline were controlled for. How well individuals coped with prodromes also predicted manic symptomology and social functioning. More individuals using stimulating coping strategies had a manic relapse, whereas those using passive coping strategies were more likely to have a depressive relapse.

There is paucity of research about the mechanisms involved in coping. Lam & Wong (1995) suggest a two-part process. The first part of coping is appraisal of the situation, prior to problem solving, or engaging available resources. This appraisal is key to the recognition of bipolar prodromes. Teaching patients how to cope with bipolar

prodromes is one element of therapy that is present in a number of approaches, such as Psychoeducational, Cognitive Behavioural, and Family Therapy. Furthermore, it is extremely difficult to establish the temporal precedence of diagnosis and poor coping skills and longitudinal studies would add to our knowledge in this matter.

2.8 Psychological Models of bipolar disorder

2.8.1 Model of behavioural activation and behavioural inhibition

The Behavioural Activation System (BAS), is described as a core vulnerability in bipolar disorder (Depue and Iacono, 1989). This model is based on a general theory of appetitive and aversive motivation originally developed by Gray (1976, 1982). BAS is conceptualised as a neural system that regulates appetitive motivation, positive affect and approach behaviour in response to cues of incentive or reward. Fowles (1987) described the BAS as “a reward-seeking or approach system that responds to positive incentives by activating behaviour” (p.418). Both conditioned and unconditioned stimuli which are perceived as rewarding activate the BAS. Depue and Collins (1999) suggest that this incentive motivated behaviour is “associated with the unipolar dimension of positive affect” (p.7).

Both Gray (1975) and Depue and colleagues (1987) have suggested that mania is tied to increased activity within the behavioral facilitation or behavioral activation system (BAS). Manic symptoms are expected to emerge when bipolar persons have high activity in this system. The BAS is conceptualized as a neural system that regulates appetitive motivation (Gray, 1975), and the dopaminergic ventral tegmental tract (VTA) is hypothesized to be

the central neurobiological substrate of the BAS system (Depue & Collins, 1999). As a motivational system, the BAS is believed to regulate a broad range of behavioural and emotional systems to facilitate responses in the context of cues of incentive. When BAS sensitivity is high, individuals are expected to demonstrate a wide range of responses to cues of incentive, including positive affect (elation and hope), increased energy, and increased psychomotor activity.

Fowles (1993) suggested that activity in the behavioral inhibition system (BIS) also influences the course of bipolar disorder. The BIS regulates avoidance behavior in response to cues of threat. Fowles suggested that BIS might limit the expression of manic behaviour.

In contrast the BIS regulates anxiety and withdrawal behaviour in response to signals non-reward or threat (Gray, 1976). The aim of the BIS is to inhibit behaviour that could lead to pain, punishment or other negative consequences. The affective component of BIS activation is thought to be negative affect (Carver and White, 1994), which promotes apprehension, vigilance, anticipation and nervousness, all of which promote escape behaviour upon threat (Fowles, 1987).

Studies of BAS in students have shown that BAS sensitivity is related to positive affect and reward seeking behaviour. In an interesting set of longitudinal diary studies Shelly Gable and colleagues (Gable et al., 2000), used hierarchical regression analyses to show that individuals with higher BAS sensitivity experienced higher levels of positive affect over time than those with low sensitivity. Furthermore, those with higher BAS sensitivity were also more likely to experience positive daily events and to rate those events as more important. She suggested that this was evidence of the approach-orientated nature

of the BAS in seeking out rewarding or pleasurable experiences. Elevated BIS sensitivity is associated with anxiety disorders and depression, whereas elevated BAS sensitivity is associated with mania, ADHD, drug abuse and alcoholism (Fowles, 1993; Fowles, 2001; Johnson et al., 2003).

For bipolar disorder specifically it has been suggested (Depue and Iacono, 1989; Depue and Collins, 1999) that BAS dysregulation is directly related to the onset and maintenance of manic mood states. This hypothesis is largely based on the phenomenological similarities between clinical manifestations of mania and extreme behavioural activation; in terms of psychomotor activity, hyperactivity, high energy and decreased need for sleep, reward incentives, desire for excitement, elated mood, and cognitive characteristics of inflated confidence and grandiosity. By analogy, depression in bipolar disorder results from an absence of BAS activation (Depue and Iacono, 1989), manifest in increased hopelessness, inactivity and lack of pleasurable activity.

Meyer and colleagues (Meyer et al., 2001) set out to test the hypothesis that vulnerability to extreme mood states in bipolar disorder is linked to BAS activity and an under regulation of that system. They found in a sample of 59 remitted bipolar I patients that BAS sensitivity was associated with manic symptoms over a six month period. They further found that BAS sensitivity was a stable and trait like feature that showed little variation over the course of their study. The same research group also showed that elevated BAS sensitivity was associated with concurrent symptoms of mania in a small non-clinical high risk sample of students with elevated scores on a measure of hypomanic personality (Meyer et al., 1999).

Johnson et al. (2000) put forward the argument that BAS activation is directly associated with the onset of mania following goal attainment events. In a study of 43 bipolar I patients they found that over the course of 2 months independent goal attainment events were predictive of manic symptoms. They argued further from these findings that with BAS activation manic patients continue to experience related symptoms that in turn promote increased reward seeking reinforcing manic characteristics that easily develop into a clinical episode of mania. Apart from reward seeking and activation of manic phenomenology in bipolar patients BAS activation is further associated with increased negative affect in response to frustration and other stressful situations (Carver, 2004), characteristics that are also often part of the phenomenology of mania.

2.8.2 Circadian and social rhythms in bipolar disorder

Generally circadian rhythm instability has long been associated with mood instability with early descriptions going back to Hippocrates (Jones, 2006; Goodwin & Jamison, 2007). More specifically daily rhythm disturbances as well as seasonal variations have been repeatedly associated with mood instability in bipolar disorder and its inherent cyclical nature (Coryell, *et al.*, 2003; Leverich *et al.*, 2003; Post *et al.*, 2003). In that circadian rhythm characteristics are directly linked to symptomatic manifestation in bipolar disorder, sleep disturbance and reduction of activity and interest in depression and a decreased need for sleep and increase in goal directed behaviours in mania. Longitudinal diary studies of bipolar patients have also showed that reduced sleep is significantly predictive of future hypomania (Leibenluft *et al.*, 1996), and sleep reduction has been described as a “common pathway” to mania (Wehr *et al.*, 1987).

In the context of a clinical approach to stabilising of social rhythm and social zeitgebers in bipolar disorder Ehlers and colleagues (Ehlers, et.al., 1988) developed a psychological treatment for bipolar disorder that is focused on the effort to regulate daily routines and daily rhythms, in terms of sleep wake cycles, regular eating and activity patterns as well as social interactions. Ehlers et al., further suggested that life events with a big impact on daily routines such as unemployment, birth of a child, etc., can cause lasting disruption in social rhythms and lead to an increased vulnerability to mania. In line with this hypothesis Malkoff-Schwarz and colleagues found in a series of retrospective studies that periods prior to manic episodes have significantly more social rhythm disrupting events compared to control periods. Furthermore, manic patients' pre-onset periods are characterised by increased disrupting events compared to the pre-onset periods of unipolar depressed patients (Malkoff-Schwartz et al., 2000).

Recent research in the circadian rhythm instability in bipolar disorder has employed actigraphic assessment of sleep and activity using wrist actigraphy (Jones, 2006). A number of these studies confirmed the association of bipolar symptomatology with activity pattern, e.g. Teicher et al., (1997) or Millar et.al., (2004). Jones et al. (2005), found that in remitted bipolar patients no sleep differences were found when compared to age matched controls, however they did find that patients had significantly less stable activity across days and more variable activity within days. The authors suggested this indicated a weaker coupling to the external environment and greater fragmentation of rhythmicity. The above evidence seems consistent with the overall instability hypothesis also suggesting that instability in bipolar individuals is not limited to acute episodes.

This suggests that bipolar individuals have an inherent vulnerability to social rhythm disturbance and dysregulation that is observable independent of current symptomatology.

In line with this vulnerability hypothesis Meyer and Maier (2006), in a study of 56 students found that students who scored highly on a measure of behavioural risk of bipolar disorders reported less regularity in the timing of activities and more variable sleep duration compared to controls.

Jones (2006) highlighted that circadian rhythm disruption is present in a range of conditions, in unipolar depression (Souetre et al., 1989), Alzheimer's Disease (Harper et al., 2005) and schizophrenia (Martin et al., 2001), but mania is not commonly reported in these groups. This highlights the importance to look at the underlying psychological mechanisms of the specific effect of circadian rhythm disruption in bipolar disorder. He argues that it seems probable that both instability of circadian functioning and a dysfunctional interpretation of circadian disruptions are critical in bipolar disorders (Jones, 2001, 2006). Developing from the earlier work of Healey and Williams (1989), Jones uses a multilevel approach to cognition and emotion to explore how circadian rhythm disruption may lead to clinical manifestations of bipolar disorder. Central to this model is the interpretation of these symptoms in a positive self-dispositional manner which increases the likelihood of positive affect and leads to behavioural responses which exacerbate the initial disruption. He described this interpretive style as the "hypomanic interpretation" and gave a theoretical account of this assertion within a multi-level model of emotion, the SPAARS model (Schematic Propositional Analogical and Associative Representation System) (Power and Dalgleish, 1997). In this model Jones integrates circadian and psychological factors in the genesis of mania and depression (Jones, 2001).

The SPAARS model suggests that there are two routes to emotion. External events are first processed analogically on a physiological or sensory level, and then processing at a propositional level introduces semantic information, this level of processing is associated

with current goals and belief systems, resulting in an overall schematic model. Emotional outputs occur via schematic and associative levels. Schematic processes include information from all other processing systems into a level "beyond verbally expressible propositional concepts" (Power et al., 1997). It is the totality of information in the schematic model which produces emotion. Thus, feedback from the analogue and propositional levels which is consistent with current goals would lead to positive affect whereas incongruent information may produce negative affect. In contrast to traditional cognitive models there is no direct route from propositional representations to emotion. These can only give rise to emotion via schematic models or through past associations between these thoughts and emotions. The associative route provides a direct link between experience and emotion through either repetition or salience. Emotional generation via this route does not require schematic appraisal to generate emotions.

In line with the SPAARS model Jones (2001) suggests that circadian disruption is integrated into the analogical stage of processing. So, the physiological (analogical) effects of increased stamina, energy and cognitive alertness may trigger an internal attribution bias and lead to hyper-positive propositional thoughts ("I am full of energy and ready to take on the world") (Jones, 2006, p.104). He suggests that on a schematic level this positively orientated (propositional) conception of the self, together with the physiological information, will be associated with beliefs about the self as powerful, others as inferior and the world as full of opportunities. It is this schematic model which is linked to mania terms of elevated mood and initiation of behaviours such as increased risk taking, reduced sleep and increased activity. Referring to the associative link to emotional experience and expression Jones further argues that any further bipolar episodes could be triggered by more minor rhythm disruptions or life events; this is consistent with the kindling hypothesis of recurrence in bipolar disorder (Post et al.,

1986; Ramana *et al.*, 1995). Mixed states in this model are explained by the conflicting or simultaneous outputs from different levels of processing.

Similarly Jones (2001) suggests that an adapted SPAARS account of depression implicates a similar internal attribution for physiological feelings. For example, slight circadian disturbance may produce the analogical effects of lethargy and fatigue. These negative symptoms, in the absence of any obvious causal factors, would be internally attributed and interpreted in terms of self-deficiency and would give rise to propositional statements, e.g. "I'm weak". This is consistent with the work of Lyon *et al.* (1999) who suggested that bipolar patients do not exhibit a "normal" self-serving bias. The negative analogical and propositional information would be appraised at the schematic level in terms of the self as ineffective or helpless, resulting in dysphoria and poor coping (e.g. sleep loss, inactivity) which prevents the natural reparation of the original disruption (Healey and Williams 1989).

Overall this model suggests that individuals suffering from bipolar disorder are more prone to circadian rhythm instability and that these disruptions are exacerbated by the initial changes and their attributions triggered by such instability (Jones *et al.*, 2006).

2.8.3 A CBT model of Bipolar Affective Disorder

Many authors have argued that there is a marked lack of a coherent psychological model of bipolar disorder (e.g. Scott 2001a; Jones 2001). Recent research, however, highlighted the role of cognitive and psychosocial factors in the development and course of bipolar disorder, and first treatment manuals were published in recent years delineating the application of CBT principles to bipolar disorders (Basco & Rush, 1996; Lam, *et al.*, 1999;

Scott, 2001b; Newman, et al., 2002). A body of research focused on cognitive factors such as attributional styles (Alloy, et al., 1999); perfectionism, deficits in problem-solving skills and elevated scores of sociotropy and autonomy (Lam, et al., 2000); and maladaptive schemata (Young, et al., 1999). These factors appear to play a significant role in the interaction of considerable changes in behaviour, reactions to and the creation of significant psychosocial stressors, disruptions in chronobiological functioning and varied responsiveness to psychotropic medications. One of the reasons for the complex pattern of factors influencing phenomenology and course of the disorder for each individual is the huge variability in the spectrum of bipolar disorders ranging from chronic cyclothymic presentations to episodic manifestations of severe depression and mania including psychotic features.

Overall, the cognitive behavioural model aids our clinical understanding of the psychopathology of bipolar disorder and the ways in which specific problems and interactions can be targeted; it does not, however, offer any further aetiological clarification of this disorder. Researchers suggested that comparable cognitive structures and biases underlie both unipolar and bipolar depression (Alloy, et al., 1999; Reilly-Harrington, et al., 1999; Lam, et al., 2000), particularly in terms of systematic attributional errors and sensitivity to personal failure or interpersonal rejection. Specific to individuals suffering from bipolar disorder is the extreme valence shift in the content of their thinking. A cognitive model of bipolar disorder needs to integrate the variability in the stress responses of individuals. In other words, depending on type of stressor or circumstance individuals with bipolar disorder can respond with the development of depressive symptoms or with the development of manic responses, this can vary between individuals or over time. The model also needs to take into account that bipolar individuals display trait like thought processes in form of long-standing predispositions

and state like responses to environmental triggers and physiological activation. Further it needs to take into account the specific effects of significant life events and environmental stressors on individuals' affect regulation, in line with a diathesis-stress model of mood disorders (Lam, et al., 1999). In particular the fact that only certain life events appear to be able to predict mania, while others do not (Johnson, et al., 1999). A clinical working model of bipolar disorder therefore needs to encompass biology, individual beliefs, and behavioural reactions, interpersonal functioning, environmental triggers and life events, and the individual's idiosyncratic conceptualisation of these events.

Beck's (1979) cognitive behavioural model suggests that depressed mood is mediated by particular patterns of thinking that accentuate mood shifts. Individuals who are depressed become more negative in how they perceive themselves, others, and the world in general, as a result they are prone to systematic cognitive distortions in that they tend to over generalise, self-blame, jump to negative conclusions, and tend to view things in black and white terms. The avoidance of social contacts and other safety behaviours often result as an interaction of mood shifts and negative thinking patterns. These cognitive styles of depression are thought to arise out of early learning experiences. Beck suggested in his cognitive model that mania is a mirror-image of depression, determined by a hyper-positive triad of self, others and the future. Scott and colleagues (2000) found that individuals with bipolar disorder demonstrated lower levels of self-esteem, overgeneralised memory, poorer problem solving skills, and higher levels of dysfunctional attitudes, particularly related to need for social approval and perfectionism. They further found that these vulnerabilities persisted between episodes in patients who were adherent to prophylactic treatment. Beck and colleagues worked on a reformulation of the original linear cognitive model for bipolar disorders (Beck, 1996; Newman, et al., 2002). This recent re-conceptualisation includes the notion of

'modes'. Modes are understood as integrated 'cognitive affective behavioural networks' of powerful combinations of schemata, overlearned behaviour pattern and intense, difficult to modulate emotions. When schemas and modes are activated by specific life events, chronobiological disruption or other such triggers, the bipolar individuals' predispositional reactions become expressed by extremities in emotional and behavioural functioning. They argue that individuals' belief system interact with their inherent perception of current stressors and events. This activation of long-standing beliefs and schemata determines their affect and behaviour, and influences their information processing by directing the individual towards information consistent with the schema. In this way a negatively valenced schema is activated during a depressed phase, directing memory retrieval towards events of loss and rejection and focusing current attention to the possibility of failure. In a manic phase, a positively valenced schema is activated, and is likely to lead to problematic decision-making by selectively ignoring the need for adaptive caution and inhibition.

Clinically, a reliable understanding of the individual's cognitive assumptions and core beliefs that encompass his or her perception of themselves, the world and the future, helps the therapist to demonstrate an accurate understanding of the individual's experiences and to focus on the assumptions and beliefs that cause most distress and dysfunction. It is therefore important to assess individuals core beliefs independent of their presenting symptom pattern. A grandiose and manic individual might have the same core schema of "unlovability" and "incompetence" as a depressed patient. Bipolar individuals appear to maintain consistent maladaptive core beliefs and schemata that shift polarity in their manifestations. The successful modification of these beliefs through cognitive therapy should result in the reduced amplification of the dysfunctional mood swings of the bipolar client.

Apart from the reformulation of the cognitive model for bipolar disorder by Beck and his colleagues, other alternatives have been formulated to capture the complex interactions between thoughts and emotions. Teasdale & Barnard (1993) differentiated between propositional and implicational "schematic" levels of information processing. They argue that propositional-level cognitions, or direct appraisals of any given information, do not directly activate emotional reactions, but are mediated on a level of implicational meaning by a process of schematic appraisal, in the context of present and past propositional information. Power & Dalgleish (1997, 1999) support this model and add an additional direct or associative route to emotions. This model has several clinical implications by disentangling the rational or schematic processes of change that appear to be primarily focused on by classical cognitive therapy approaches from the associative or direct associations of certain cues and emotional reactions. Jones (2001) utilised this multilevel approach to emotion and cognition to investigate the vulnerability of bipolar individuals to mood changes following disruptions in their circadian rhythms. Following this model in individuals with bipolar disorder schema change is achieved through associative links through behaviour modification and corrective experiences, rather than rational cognitive techniques such as the challenging of automatic thoughts and restructuring. Patients should therefore be encouraged to experience subsyndromal mood changes and stimulation utilising adaptive coping strategies with the subsequent absence of prodromal symptoms.

2.9 Summary

To different degrees, the psychosocial factors discussed here have all been found to have some influence on the course of bipolar illness. Each individual patient will have his or her own particular set of factors influencing outcome, coupled with an idiosyncratic set of risk factors making the disorder as a whole a complex one to treat. A couple of themes have permeated the discussion of risk factors, one being the difficulty of studying so called euthymic patients. The criteria for the length of time individuals must have been euthymic differs from study to study. Secondly, the methods used to assess psychosocial phenomena vary, again making it difficult to compare studies. Nevertheless, psychosocial factors have consistently been shown to shape various outcomes in bipolar disorder, with some studies (e.g. Hammen et al., 2000) finding that psychosocial factors were significantly stronger contributors to outcome than psychiatric symptoms, frequency of episodes and hospital admissions.

More research is required looking at the delineation between those factors influencing outcome and those that are risk factors for the disorder. Alloy (1999) suggests that the criteria for a risk factor are as follows: 1) it must temporally precede mood episodes or symptom exacerbations and 2) it must exhibit some stability independent of symptomatology. Kleindienst et al. (2005), in a systematic review of 9 psychosocial and demographic variables believed to be related to outcome under lithium, found that high social status, social support, compliance and dominance were protective factors, whereas stress, high EE, neurotic personality traits, unemployment and high number of life events were risk factors. It is also important, particularly in the study of psychosocial factors that measures are taken to ensure that studies are methodologically sound. In particular, more longitudinal studies with an appropriate sample size and using validated measures

are needed. Researchers must give tentative conclusions with regards to direction of causality unless studies satisfy certain criteria. An understanding of risk factors and factors influencing outcome would assist the clinician in the selection of appropriate therapy, at the appropriate time.

Understanding when therapies are effective i.e. what cycle of the disorder is the most important time for intervention, is essential. Little focus has been given in the literature to the optimum time to administer the interventions described in the first section of the review. Timing of interventions is also complementary to the idea of combinations of therapies. It may be that when a patient is euthymic and more receptive then psychoeducation may be appropriate, in order for the information to be better absorbed and understood. When the patient is experiencing an acute episode of depression it may be more important to provide CBT. Some studies show that patients suffering from a mild depressive episode can usually gain benefit from psychoeducation as well (Michalak et al., 2004), but the presence of (hypo)manic symptoms limits its impact (Rouget & Aubry, 2007). Indeed in the 2-part Life Goals Program, de Andrès et al. (2006) found that participants who had higher depression scores at inclusion were more likely to continue with the second phase of the program. They suggest that patients with higher depression scores are more motivated to seek help. Bieling et al. (2003) further suggest that individuals suffering from bipolar disorder and borderline personality disorder would benefit from psychotherapy not just as a maintenance phase adjunctive treatment but also in the acute phase of treatment. CBT and IPSRT have been identified as being useful in reducing subsyndromal mood symptoms and aiding functional recovery (Zaretsky, 2003). The growing evidence showing a relationship between residual symptoms and prodromal symptoms (Fava, 1999), and shortened time to relapse (Perlis et al., 2006), signal the value of identifying these symptoms and intervening at this level (Watkins, 2003).

Basco et al. (2007) propose that there is no absolute rule for when to begin psychotherapy, although it is generally accepted that patients need to be relatively free of intense psychotic symptoms and stable enough to tolerate interaction without becoming agitated or irritable. In the early stages patients may be too ill to benefit from or tolerate therapy. However they suggest that during this early phase, useful work can be done with families. Falloon (2003) concurs that early family-oriented intervention when mood symptoms first emerge may prove successful in preventing major episodes, associated social morbidity and potential suicide risk.

As mentioned frequently throughout the review, it is only recently that studies have begun to look at different groups and subtypes within the bipolar spectrum. Often exclusion criteria exclude the more chronic and difficult to treat patients from studies e.g. patients with comorbid disorders, rapid cyclers, the elderly and the young. This has left us to question whether the interventions examined are only effective in the less serious cases. However studies on subgroups are now emerging, and research programs are beginning to find results with more clinically relevant samples. It could be that different therapies and psychosocial factors play differing roles across the bipolar spectrum. Certainly it would appear that future interventions will be tailored and subtype specific rather than generic therapies.

Despite largely trying to focus on a fairly homogenous (albeit largely unrepresentative) sample, conclusions drawn from the studies described above are still based on the collective data from groups of diverse individuals. Bipolar disorder covers a broad spectrum of symptoms and individuals suffering from the disorder can vary in the range of symptoms they experience and their severity. Although all will share diagnostically similar

fluctuations in mood, the illness itself has an idiosyncratic manifestation. In the studies examined in this review, individuals may share a common diagnosis, but they may vary considerably in other features that underlie and characterise their distress e.g. behavioural patterns, life circumstances, coping skills, comorbid conditions, and chronicity of problems (Henin et al., 2001). This variability should be taken into account and individuals ought to be treated as unique. Henin et al (2001) acknowledge this and propose flexibility strategies in the application of cognitive behavioural treatment so that the therapist can individualise therapy sessions around the issues specific to each patient while maintaining a reproducible format for treatment.

It has also been suggested that specific elements of the therapeutic process or characteristics of the patient are responsible for a good outcome e.g. the setting of the therapy, motivation (Leff et al., 1989), method of therapy: discussion versus behavioural rehearsal (Tarrier et al., 1988). Future research could perhaps concentrate on narrowing the gap between experimental and comparison conditions, in order to find the essential active ingredients (Goldstein & Miklowitz, 1995).

Emphasis is being drawn away from the efficacy of a treatment being measured by changes in symptom levels, relapse rate and hospitalisations. The research to date has shown that therapies, be they pharmacological or psychological, have been inadequate in eradicating symptoms permanently or preventing relapse indefinitely. All of the therapies mentioned have shown some evidence in reducing time to relapse. Therefore striving for therapies that don't appear to exist rather than focusing on refining the therapies that we have may be wasteful. Combining therapies such as has been done with IFIT appears to be promising in terms of increasing time to relapse and reducing depressive symptoms (Miklowitz et al., 2003).

Measures of outcome should reflect improvement or decline appropriately. When working with psychosocial interventions it appears more appropriate to consider outcome through changes in the variables that the intervention focuses on, for instance family therapy producing changes in EE. However changes in these psychosocial variables need to show changes in the outcome for the individual. However symptom severity and relapse are inappropriate markers for outcome and recovery in bipolar disorders. There is a need for measurement of outcomes which reflect clinical realities (Geddes & Goodwin, 2001). A more valid outcome and one that reflects reality for individuals with bipolar disorder is tangible changes in quality of life. Quality of life has been increasingly highlighted as an important outcome measure for psychosocial intervention in bipolar disorder, in looking at quality of life we can analyse quantifiable changes in individuals' perceptions of well being and functioning as a consequence of their illness (Robb et al., 1997). Reducing the impact that repeated episodes have upon domains of functioning and improving inter-episode functioning appears to be a sensible and more appropriate route to approach treatment of bipolar. This is the major focus of psychosocial intervention.

III Psychological Interventions for Bipolar Disorders

3.1 Scope for psychological intervention & preventative strategies

Pharmacotherapy remains the first-line and mainstay of treatment for bipolar disorder, used to treat symptoms in the acute, stabilisation, and maintenance phases of the disorder (Miklowitz & Otto, 2006). However psychological interventions have seen an increase in interest in the last 20 years and now appear in recent expert recommendations such as the NICE Clinical Guidelines 38 (National Institute for health and Clinical Excellence, 2006), the SIGN Guidelines (Scottish Intercollegiate Guidelines Network, 2005), the Canadian Network for Mood and Anxiety Treatments (Yatham et al., 2005) and the American Academy of Child and Adolescent Psychiatry Practice Parameter (McClellan et al., 2007).

Psychological interventions are largely used as adjuncts to pharmacological treatment. Their publicised role has been to improve treatment outcomes by increasing medication compliance. Indeed the bulk of the literature to date relates to studies of compliance augmentation. Lithium remains the drug of choice for pharmacological treatment of bipolar disorder. However compliance of individuals maintained on lithium is low (Jamison et al, 1979; Scott & Pope, 2002), side effects are prevalent, and efficacy is questionable. Relapse and sub-syndromal symptoms often continue even with regular medication use (Gitlin et al., 1995; Maj et al., 1996). In one study 20% of patients maintained on lithium continued to experience moderate or severe impairment (O'Connell et al., 1985). Another found that patients taking lithium after a manic episode

did not differ in symptomatology or overall adjustment to those who did not (Harrow et al., 1990). Recent evidence has also suggested that medication compliance is unrelated to relapse (Yan et al., 2004) suggesting efficacy of psychosocial interventions may be based on different mechanisms. Given these problems with non-compliance, high relapse rates, side effects, and residual symptoms, an ample need for additional treatment strategies has been recognised (Otto et al., 2005).

This review examines the development and rationale of a range of psychological therapies and the outcomes of studies that have examined and validated them. The review will explore Psychoeducation, Cognitive Behavioural Therapy, Family Therapy and Inter-Personal Therapy/Inter-Personal and Social Rhythm Therapy.

3.2 Assessment & psychological formulation

3.2.1 Psychoeducational approaches

Psychoeducation occupies a key position in psychological approaches to bipolar disorder (Rouget & Aubry, 2007; de Andrés et al., 2006). Qualitative research has suggested that individuals suffering from bipolar disorder have been found to have reduced educational attainment (Glahn et al, 2006) and major educational needs (Pollack, 1995). These needs range from basic understanding of the disorder to how to live effectively in society and relate to self and others.

Fundamentally, psychoeducation provides the patient with information about their illness. In a broader sense, it aims to provide patients with strategies to improve their illness

management skills “through the bidirectional sharing of relevant information” (Callahan & Bauer, 1999). Indeed a recent psychoeducational program ‘The Life Goals Program’ (Bauer & McBride, 2003) claims to go “beyond one-way information transfer and stimulates active self-management and collaborative activities” (Bauer et al., 2006a). Such illness management may include identifying symptoms and implementing relapse-prevention procedures (e.g. emergency medication); promoting drug adherence (Gonzalez-Pinto et al., 2004); minimising risk factors (e.g. substance abuse, interpersonal stress); maximising protective factors (e.g. regular sleep/wake cycles) (Miklowitz & Johnson, 2006); diminishing stigma; and increasing acceptance of the illness (Zaretsky, 2003). It involves a series of sessions built around a biopsychosocial medical model. This provides a theoretical framework for the patient that helps them build a practical approach to coping with the disorder (Colom et al, 1998).

The effects of psychoeducation have been investigated in several studies and in various formats: individual; group; with partners; and in families. Studies which examine psychoeducation in the context of the family will be discussed under Family Therapy, since this is the focus of the intervention. In some studies psychoeducation is delivered alone, whilst in others it is administered in combination with other therapeutic approaches. Almost all cognitive-behavioural approaches have a psychoeducative element to them. Two recent studies have incorporated psychoeducation into a multicomponent intervention program (Bauer et al., 2006a, b; Simon et al., 2006). Traditionally psychoeducation focuses on interventions in times when the individual is euthymic, in order to ensure a better assimilation of the information bestowed (Vieta, 2005; Rouget & Aubry, 2007). However not all studies take this approach.

Early trials focused upon simple education, with the level of intervention varying between studies. One study provided a package including a videotaped lecture, a written handout providing information about lithium and a follow-up home visit to 30 outpatients in remission. They were compared to 30 controls who received lithium treatment alone. Increases in knowledge surrounding lithium were found (though these didn't reach significance) and significant increases in positive attitudes to medication adherence; with the intervention group having significantly better adherence to lithium (Peet & Harvey, 1991). This method of measuring efficacy has been criticised by other researchers for being indirect (Colom et al., 2003a).

Conversely, a retrospective study of 43 individuals in group psychoeducation did not find any difference in medication adherence, but did find a reduction in symptomatology, relapse, hospitalisation, and increased social functioning (Cerbone et al., 1992). The facilitators in this group were also providing medical management, which could have decreased the response time to prodromes. The fact that medication levels did not change significantly before and after the group suggests that compliance is not necessarily linked with good outcome, and perhaps psychoeducation has greater benefits than simply improving outcome by increasing medication compliance. However the retrospective nature of the study suggests caution in the interpretation of results.

Psychoeducation was also investigated in a group format by Bauer and colleagues (1998) who created the Life Goals Program (Bauer & McBride, 1996). This intervention included both Bipolar I and II patients. The two aims of the program were to increase participation in the medical model by providing education on the disorder, and to assist the patients to achieve social, occupational or leisure goals that have not been reached due to disruption caused by the disorder. It also included identification of individual prodromes and

adaptive coping strategies. This initial pilot found that the program increased knowledge about bipolar disorder and that around half of the original sample achieved their goal.

Following the release of a French version (Bauer & McBride, 2001), The Life Goals Program was used in a specialised bipolar outpatient clinic in Switzerland. An open study was conducted by de Andrés et al. (2006) with the main focus to measure patient satisfaction with the program. Altogether 45 bipolar patients entered phase 1 of the program which consisted of 6 highly structured psychoeducational sessions; and 36 patients completed phase 1 and of those 17 entered phase 2 which was a year-long behaviourally structured program focused on goal identification. Over 80% of patients said they were 'very satisfied' with the information delivered during phase 1. After attending phase 2, patients reported subjective improvements in mood stability, relapse prevention strategies, and coping with relapse. However this was an open study and with no control group the results are difficult to draw clear conclusions from.

Learning to identify early warning signs of affective change is a specific aspect of psychoeducation, not always included in the early trials. It allows idiosyncratic profiles to be developed often including aspects such as sleep, appetite and interpersonal disturbances. These profiles can then be monitored by both the individual and the family. A study by Perry and colleagues (1999) in a controlled trial taught individuals currently in remission to identify early warning signs and seek prompt treatment. It focused on time to relapse and changes in social functioning to assess the efficacy of the intervention. They were successful in increasing time to first manic relapse but not depressive, possibly due to manic prodromes being easier to observe than depressive. Manic early warning signs (EWS) are thought to be qualitatively different from other interepisodic symptoms and fairly consistent from one episode to the next. Meanwhile, depressive EWS are

qualitatively similar to other interepisodic symptoms, but are more severe and frequent (from Morriss symposium – original source not cited). Furthermore the onset of mania is typically more acute than depression, which develops more insidiously, which may help explain why manic prodromes are easier to recognise. Altman et al. (1992) found that conceptual disorganisation is a key prodrome for depression, which may be difficult to identify in oneself. In addition, without the skills to cope with depressive episodes, greater awareness is ineffective. In the Perry et al. study, significant improvements were found for social functioning and employment which may be related to greater feelings of self-efficacy when coping with relapse. Anti-depressant use increased in the experimental group. This further suggests that medication compliance is not a relevant outcome for psychological treatment, as individuals may, through increased awareness, seek out additional medications.

Although increasing adherence to drug treatment is often a goal of psychoeducation, it can also be a confounding factor in assessing the efficacy of psychoeducation. It is difficult to determine what the underlying mechanism of change really is. Colom et al (2002, 2003a,b) proposed that psychoeducation goes further than simply improving outcome by increasing medication compliance. The authors were interested in why patients keep suffering relapses even when they follow their prescribed drug treatments.

Colom et al (2002) assessed the usefulness of psychoeducation in fully compliant patients, therefore controlling for the effects of drugs. 50 bipolar patients were randomised into two groups, one receiving psychoeducation (N=25) and the other not (N=25). Both groups also received naturalistic pharmacological treatment but no further psychological treatment was given. At the end of the two-year follow-up, 60% of patients in the intervention group had relapsed compared to 92% in the control group. The number of

relapses and the number of depressive episodes were also significantly lower in the intervention group. Psychoeducation seemed to show its efficacy in preventing relapses beyond the enhancement of drug treatment adherence. The authors theorised that the action of psychoeducation "may lay on a tripod model composed by lifestyle regularity and healthy habits, prodromal signs detection and early intervention and treatment compliance."

The main limitation of the above study was the small sample size, so the same researchers followed this study up with a larger randomized controlled trial of a structured group psychoeducation intervention (Colom et al. 2003a). They compared 60 currently euthymic patients with Bipolar I and II receiving structured group psychoeducation combined with standard psychiatric and pharmacologic treatment with 60 patients participating in weekly non-structured group meetings rather than psychoeducation. Again patients were followed up for 2 years. With recurrence as the primary outcome measure, during treatment 60% of control group relapsed compared to 38% of the treatment group. This difference was significant and was maintained to 12 month follow-up. They also found the treatment group had significantly greater time to relapse. Unlike other studies which found interventions only to be protective of manic and hypomanic episodes (Perry et al, 1999), this intervention was found to protect against all polarities of episode. This trial included teaching on the detection of prodromes and lifestyle regularity, the latter of which has been developed through Interpersonal and Social Rhythm Therapy and will be discussed later. Including the social rhythm aspect may have had a protective effect against depressive episodes that pure psychoeducation and teaching of prodromes did not.

Colom and colleagues (2003b) followed the above studies with a further exploration of the mechanisms of psychoeducation. In a single-blind, randomised, prospective clinical trial they investigated whether the impact of psychoeducation was simply based upon increased medication compliance. The patients were 50 euthymic Bipolar I individuals who were fully compliant to their medication regimes. The psychoeducation regimen was the same as the above study; the patients were followed up for 2 years on a monthly basis. The primary outcome measure was recurrence. During the therapy and at the end of the 2 year follow-up the psychoeducation group experienced significantly fewer manic and depressive recurrences. The findings again indicate an effective role for psychoeducation above and beyond compliance. The authors suggest that the detection of prodromal syndromes may play the most important role. The sample used was however very severe and complex therefore there may be generalisation problems.

The relationship between adherence to lithium and treatment attitudes and knowledge was examined more recently by Rosa et al. (2007). 106 bipolar patients participated in monthly psychoeducational groups. The package of psychoeducation included written information about the disorder, monthly meetings with a psychiatric nurse, occasional discussions with invited speakers and encouragement to join the local bipolar disorder patients association. They found that level of knowledge about lithium and bipolar disorder was directly correlated to treatment adherence. They proposed this was because psychoeducation facilitates acceptance of the disease and maintenance therapy. This is a view shared by Strauss & Johnson (2006) in their study examining the role of treatment alliance in the management of bipolar disorder. They also found that stronger treatment alliances predicted less negative attitudes about medication and less stigma about the disorder.

Trials of psychoeducation in bipolar disorder appear to be limited across various contexts. Most studies involve patients with bipolar I disorder and trials including patients with comorbid disorders, different subtypes, rapid cycling, and the young and old populations are rare. Nevertheless, the benefits of psychoeducation have been extended to bipolar patients with personality disorder in at least one study. Colom et al (2004) randomised individuals with Bipolar I and any personality disorder to either group psychoeducation or a non structured intervention. Recurrence, time to relapse and time spent in hospital was significantly lower for those in the psychoeducation group. This is a promising result, albeit with a small sample, given that the intervention was not altered to focus on the specific needs of this group.

In most of the above studies there has been an emphasis on medication compliance, symptom measures and recurrence rates to determine efficacy of the intervention. Nevertheless, some researchers have started to highlight that effective treatment may be more importantly measured on the basis of functional recovery and not on symptomatology. Michalak et al. (2005) assessed the impact of a time-limited psychoeducation group therapy upon perceived quality of life (QOL) among bipolar patients. Altogether 57 euthymic or mildly symptomatic bipolar patients took part in a standardised 8-week group psychoeducation course and completed QOL questionnaires at baseline and at the end of 8 weeks of therapy. The group found that bipolar patients continue to show impaired quality of life even when euthymic, highlighting the need for interventions to target this issue. In particular recent depression was significantly associated with lower baseline scores, in keeping with another study (Ozer et al., 2002). Group psychoeducation was associated with an increase in quality of life scores, with two particular domains (physical functioning and general satisfaction) increasing significantly. This increase in QOL is perhaps more striking when one considers that the sample were

relatively asymptomatic at baseline, and although still showed impaired QOL, may have been less likely to exhibit large change scores. Thus the potential impact of psychoeducation on quality of life may be underestimated. However this study was not without considerable limitations. Retrospective chart review was used to help assess treatment effects, there was no control group for comparison, and medication was not controlled for.

There appears to be a growing trend for psychoeducation to incorporate elements of other therapies, or to be part of larger more integrated therapies. Such multicomponent programmes are discussed later in this review. Bernhard et al. (2007) examined the efficacy of a psychoeducational programme integrating cognitive behavioural elements. 40 bipolar patients were randomised into an intervention group or waiting list control. 3-month follow-up data showed that the intervention group did significantly better than the control group on all variables (knowledge of the disorder, symptoms, social functioning, quality of life, medication compliance), although only trends rather than statistically significant results were noted. Perhaps, if the study follows the tendency of other studies to show increasingly significant results over a longer time period, the data at 6, 9 and 12-month follow-up, yet to be published, will yield more significant findings.

3.2.2 Cognitive therapy approaches

Early investigations of CBT techniques in bipolar disorder focused almost solely on the adherence to medical treatments. Main examples of this particular CBT application are Benson 1975 and Cochran 1984. Benson (1975) reports a retrospective analysis of 31 bipolar disorder patients who were all in a manic phase at the start of treatment, receiving a combination treatment of Lithium and psychotherapy. Comparisons were

made between relapse in this group of people with a diagnosis of bipolar disorder and previous reports of relapse rates with Lithium alone. He reports that 14% of his patients relapsed compared with reported mean relapse rate of 34% with Lithium alone. He suggests that psychotherapy is important to keep the patient motivated to continue Lithium, to provide basic therapeutic support and to monitor patient's mood as a way of early detection of falling serum lithium levels. Cochran's (1984) study is probably the most cited paper in the context of cognitive therapy for bipolar disorders. She evaluated the effectiveness of a preventative treatment adherence intervention with 28 outpatients with a diagnosis of bipolar disorder who have recently started Lithium treatment. The intervention consisted of six sessions of a modified cognitive-behavioural treatment aimed at cognitions and behaviour that seemed to be interfering with treatment adherence. Comparison was drawn with a control group who received standard outpatient follow-up, at the end of treatment and after 6 months follow-up. Neither the patient self-report nor the lithium levels showed an effect of the intervention, solely the psychiatrists' observation showed better perceived adherence in the treatment group after therapy. At 6 months follow up, patients in the treatment group showed significantly less hospitalisations and affective episodes. The intervention as described does not seem to take into account symptoms and other manifestations of the disorder, but only pays attention to compliance with pharmacological treatment.

A number of studies since focused predominantly on relapse prevention and the identification of prodromal symptoms and early signs of relapse. Perry and colleagues (1999) investigated 69 patients with a diagnosis of bipolar disorder who had had a relapse in the previous 12 months. Subjects were randomised into two conditions; 7-12 sessions with a research psychologist plus routine care or routine care alone. The CBT intervention consisted of teaching patients to recognise early symptoms of manic and

depressive relapse and producing and rehearsing an action plan. By comparison the treatment group experienced significantly longer intervals until manic relapse than the control group. They further found significant improvements on measures of social functioning and employment in the treatment group compared with the control group 18 months after the baseline assessment.

Several more comprehensive studies utilising a CBT framework focused not only on treatment adherence, relapse prevention, and reduction of symptomatic distress but also on psychosocial functioning. Palmer and colleagues (1995) describe a psychoeducational and CBT programme in a group format for people with a diagnosis of bipolar disorder, currently in remission. Four participants attended 17 weekly group sessions. At the end of treatment 3 out of the 4 participants showed significant improvements in depressive and manic symptoms. Three out of the 4 of the participants showed significant improvement in their social adjustment at end of treatment and 2 at follow-up. Zaretsky and colleagues (1999) designed a cognitive behavioural intervention focusing on the treatment of acute symptoms rather than relapse prevention. They demonstrated, in a matched case controlled design, the effectiveness of a 20 session CBT intervention for acute depression in the context of a bipolar disorder compared to the effectiveness in recurrent unipolar depression by comparing both groups in parallel. They found that depressive symptoms in eight bipolar and eight unipolar patients were significantly reduced after CBT intervention. Lam and colleagues (2000) describe a cognitive therapy approach for a total of 12 bipolar patients. The treatment consisted of 12-20 sessions over six months. On a global symptom level (over 12 months) the treatment group had significantly fewer episodes and fewer hospitalisations compared to the control group. The monthly self-report and observer ratings of manic and depressive symptoms confirmed that there was significantly lower level of manic and depressive symptoms in

the treatment group over the course of the 12 months. The therapy group performed significantly better on medication compliance, social functioning, self-controlled behaviour and coping with mania and depression prodromes. Patelis-Siotis and colleagues (2001) reported outcomes of a 14-session adjunctive group cognitive behaviour therapy treatment for patients suffering from a bipolar disorder. 49 outpatients with a diagnosis of bipolar disorder currently maintained on a stable mood level on medication treatment participated in a CBT group therapy programme focusing on psychoeducation and cognitive behavioural intervention strategies. The results indicate no significant changes in mood related symptoms between baseline and end of treatment, however, they found a significant increase in psychosocial functioning. Scott and colleagues (2001a) report outcome of a randomised controlled study testing the feasibility and potential benefits of cognitive therapy for people with a diagnosis of bipolar disorder. Following assessment, patients were randomly assigned to immediate cognitive therapy or 6 months waiting list control condition. Both groups contained 21 subjects. Patients were followed up at 6 monthly intervals for a maximum of 18 months. In comparison with the waiting list control groups the CBT group showed significant reductions in symptoms and improvement in global functioning. They also found that significantly fewer subjects met criteria for relapse after CBT than before and hospitalisation rates were significantly lower in the year after CBT intervention.

Cognitive Behavioural Therapy (CBT) for unipolar depression has been adapted for illnesses such as schizophrenia and bipolar disorder. The advantages of Cognitive Behavioural Therapy over other models is that it works on dysfunctional interpretations and underlying dysfunctional beliefs, in tandem with learning new skills to help prevent relapse (Scott, 2001). New skills may be recognising and dealing with psychosocial stressors, increasing coping skills or learning new strategies to deal with cognitive or

behavioural problems. Some controlled trials of CBT for schizophrenia have resulted in a reduction of psychotic symptoms (e.g. Tarrier et al., 1998). Others (e.g. Barrowclough et al, 2006) found that whilst group CBT is less likely to have an impact on hallucinations and delusions, it may have important benefits including feeling less negative about oneself and less hopeless for the future. The authors stress the importance of these changes in view of the role that hope and empowerment have in models of recovery (e.g. Resnick et al, 2005). With a focus on improving psychosocial function, a single case study by David & Lysaker (2005) showed CBT to be effective in improving work behaviours and symptoms of emotional discomfort in a patient with schizophrenia. Studies of CBT in unipolar depression have led to prevention of relapse (Evans et al., 1992; Bockting et al., 2005) and increased functioning (Ravindran et al, 1999). Research also uncovered the relatively long-term effect of cognitive therapy in preventing relapse in depression compared with medication as an acute treatment (Evans et al, 1992).

Since then CBT strategies have been developed specifically for bipolar disorder. Most cited in the literature is the pioneering work of Basco & Rush (1996) who developed a cognitive-behavioural model proposing that changes in mood and cognition in bipolar disorder are accompanied by behavioural changes, which in turn have a negative impact on the individual's psychosocial functioning. An example of this is an individual's mood shifting towards depression. A typical change in the process of cognitive functioning may be a decrease in speed of thought and in content it may be an increase in negative thoughts. These changes may be accompanied by behavioural changes such as a decrease in activity. These changes may, in turn, have an impact on the individual's psychosocial functioning such as neglect of family responsibilities, or slowed work functioning. Stress related to these psychosocial problems can cause symptoms to worsen and as symptoms further alter functioning, new stressors are consequently created. Based upon this model

Basco & Rush (1996; 2005) developed CBT to target cognitive, affective, and behavioural changes in depression and mania. The main components of the treatment are: (1) expanding patient's knowledge about the disorder; (2) development of an early warning system that allows them to anticipate the onset of a mood swing and respond accordingly; (3) management of the illness e.g. improving and maintaining adherence to drug treatments, psychosocial interventions, and lifestyle changes. Their approach however has never been subjected to a systematic study of its efficacy or effectiveness.

More recently Brondolo & Mas (2001) developed a cognitive behavioural program designed expressly for improving medication adherence in bipolar disorder. The therapy consists of behavioural, cognitive, and interpersonal interventions and focuses on three goals: prevention of suicide; mood stabilisation; and achievement of individual personal and occupational goals. In more depth, the behavioural interventions include: goal setting, which is a collaborative process providing an agenda for treatment; task analysis, which is used to identify the skills needed to achieve a particular goal and the symptoms which might impede the realisation of that goal; and self-monitoring, which helps the patient recognise their symptoms and the impact the symptoms have on their functioning. Together these interventions provide the framework for therapy. Cognitive interventions are aimed at addressing the emotions and feelings e.g. shame, defensiveness and fear, which can hinder treatment. Such interventions include identifying and evaluating maladaptive or unhelpful patterns of thinking and addressing patients' misconceptions about their illness. Finally the interpersonal relationship between the patient and therapist provides the context in which the interventions take place.

Like the early psychoeducation studies, early studies of CBT were weak in their methodology, lacking controls and with small sample sizes. Statistical significance of

findings was at times left undiscovered. The earliest study of CBT for bipolar disorder was carried out by Cochran (1984). It was a 6-week short-term preventative approach to aid medication compliance of patients treated with lithium. The premise is that dysfunctional attitudes and beliefs inhibit compliance. The author adapted a cognitive-behavioural approach that had been developed for depression (Beck et al. 1979). During the sessions the therapist worked to help alter cognitions and behaviours that disrupt compliance, through developing an understanding in the individual that thought affects behaviour, while encouraging more adaptive thinking. Participants in the intervention group had better composite adherence scores (as assessed by medication compliance, appointment attendance, and lithium levels) than the control group during treatment and at 6-month follow-up. The intervention group were also significantly less likely than controls to have major compliance problems, terminate lithium treatment against medical advice and to be hospitalised. This treatment in this study employed specific cognitive and behavioural techniques, rather than using case formulation (Gutierrez & Scott, 2004). It has been argued that the intervention was psychoeducational rather than cognitive in nature (Zaretsky, 2003). Furthermore the issue of using compliance as a measure of efficacy is debatable.

Palmer & Williams (1995) were the next to show interest in CBT. They carried out an exploratory study with 4 participants in conjunction with pharmacological treatment. All participants received 17 weekly group sessions of 90 minutes duration with 6 follow-up sessions monthly. Outcome was measured more directly than the Cochran study through changes in symptom levels, NHS resources used and social functioning. There was also a shift in focus away from medication compliance with increased focus on early identification of changing mood states and the development of coping strategies at these times. As the outcomes were considered separately for each individual and there was no

control group, conclusive findings are difficult to draw. However the benefits of group therapy for sharing ideas and support were expressed.

Satterfield's (1999) case study of cognitive-behavioural therapy for a rapid cycling bipolar case compared the individuals' symptomatology when being treated with pharmacology alone to when he was treated with both pharmacology and CBT. The rationale behind the intervention was based upon the disruption of biological rhythms by stress, life events or disrupted social rhythms or 'zeitgebers' (Ehlers et al., 1988). This disruption in biologically vulnerable individuals is thought to lead to biological deregulation which in turn can form a bidirectional relationship with the above factors initiating a self-perpetuating cycle. The therapy focused on the prediction, prevention, and treatment of affective episodes and areas covered included early warning signs profiles, protective behavioural and cognitive coping mechanisms, social regularity and increased autonomy. Although the author failed to investigate statistical significance, he stated a decrease in frequency and intensity of affective episodes, decreases in hopelessness and anxiety, and an increase in global functioning.

Very few other studies have looked at CBT for rapid cycling bipolar disorder, despite this group being particularly in need of effective, adjunctive treatment. Reilley-Harrington & Knauz (2005) present a further case study illustrating this treatment approach and describe specific obstacles and considerations in treating this challenging population.

Cognitive Behavioural Therapy can be applied during different phases of bipolar illness, although many studies indicate that efficacy is higher during periods of euthymia, and for bipolar depression rather than mania. Studies of CBT have not been limited to remitted individuals and trials have included individuals in various phases of the illness. Indeed

some authors feel that the short-term nature of CBT interventions makes them appealing for treating patients in acute episodes (Basco et al., 2007).

Zaretsky et al. (1999) carried out an early pilot trial of individual CBT modified for bipolar disorder (Basco & Rush, 1996), combined with concurrent treatment with mood stabilising drugs. They compared individuals with bipolar disorder who were in the depressive phase to those with unipolar depression, also currently depressed. They found a significant decrease in pre- to post- scores on the Beck Depression Inventory for both groups. They did not find any significant changes in the underlying dysfunctional attitudes of the bipolar group, suggesting that more intensive treatment is required. It is also suggested that work be continued into euthymic periods when the individual is more receptive. The study was limited however by its small size which may account for the findings, and for its lack of a bipolar control. There was no assessment of changes in functioning.

Lam and colleagues (2000) investigated the outcome of CBT for those currently in remission. A pilot study of individualised CBT consisted of 12-20 sessions of CBT for Bipolar I individuals, not in acute episode. The treatment group showed significantly fewer bipolar episodes, less manic and depressive symptoms at 12-month follow up, better coping strategies for manic and depressive bipolar prodromes, and increases in social functioning. This study controlled for medication level to ensure that the effects seen were not a result of increased medication prescription. There were, in fact, significantly less neuroleptics prescribed in the therapy group for the 6 months post-therapy.

A small open trial conducted by Fava et al. (2001) of 15 remitted bipolar I patients with a 2- to 9-year follow-up found a significant reduction in residual symptomatology which is thought to increase vulnerability to future episodes (Fava et al., 1999). This provides some hope for the lasting effect of cognitive-behavioural strategies on symptoms in Bipolar Disorder. However, without a control group it is impossible to say whether the positive effects were due to the therapy itself.

A further randomized controlled study of cognitive therapy carried out by Lam et al. (2003) in remitted individuals showed positive results with significantly fewer individuals in the treatment group experiencing depressed, manic or mixed episodes; and when these episodes are experienced they were of shorter duration and with decreased chance of admission. The cognitive therapy group also had higher social functioning. Additional follow-up data presented after 2 years (Lam et al, 2005a) showed that the effect on relapse was most marked in the 6 months of therapy and the following 6 months, and there was no significant effect over the last 18 months. Effects on relapse were significant on depression only, but it was found that during the 30 month period of the study, individuals continued to have shorter bipolar episodes. In addition, the experimental group had better coping strategies for dealing with manic and depressive prodromes at month 24 of the study. Goal attainment was reduced, which the authors identified as a vulnerability factor for future relapse. The increase in social functioning lasted until the 2 year follow-up, falling to below significance after this.

A group CBT study carried out by Patelis-Siotis et al. (2001) focused on the premise that bipolar disorder results in significant impairments which move beyond those expressed in symptomatology, and that are apparent in interepisode periods. They highlight a delay in functional recovery following treatment and suggest that it is essential to target

treatments at these periods. 49 bipolar I and II mildly depressed or euthymic individuals took part in this open trial. There were significant improvements in the psychosocial functioning of the individuals but no change in symptomatology. However this is perhaps not surprising given that the patients were relatively asymptomatic at baseline. Due to limitations of the study, for instance a lack of control group, it is difficult to decipher whether findings were a result of the CBT or the group format. Low level of symptoms in the treatment group may also have masked any changes. Nevertheless the results still suggest that group CBT interventions may be of value for improving quality of life and maintaining symptom stability.

Scott et al (2001) carried out a pilot study of CBT with a focus on relapse prevention. 21 bipolar I and II individuals in all phases of illness received cognitive therapy immediately, with a further 21 acting as a waiting list control. The inclusion of patients with comorbid personality disorders and histories of medication non-compliance made this a more clinically relevant group of patients compared to other studies. The treatment was broken down into education regarding bipolar disorder and introduction to the cognitive model of bipolar disorder, with individualised formulations. Cognitive and behavioural approaches to management of symptoms and dysfunctional attitudes were then introduced. Unlike the Patelis-Siotsis and colleagues' (2001) study, Scott et al. found a significant decrease in symptomatology in the intervention group after 6 months of CBT. There was also a significant improvement in functioning, with a tendency for symptoms and functioning to deteriorate immediately after therapy. Relapse rates showed a significant reduction at 18-month follow up, with depressive relapse decreasing the most. Scott and colleagues warn that while cognitive therapy for bipolar disorder helps individuals manage their early warning signs, it will not prevent episodes from occurring. Interestingly, after 6-months observation, the control group were then given CBT to

assess within-person change. This group also showed similar improvement in symptoms and functioning from pre- to post-CBT and had significantly fewer hospitalisations in the 12 months post-treatment compared to their 6-month waiting list period.

However, in a more recent RCT, Scott et al. (2006) were unable to replicate these positive findings. They compared adjunctive CBT to treatment as usual in 253 participants across five treatment sites. This time no significant differences were found between the groups in recurrence rate, medication adherence, or symptom control. Further analysis found that CBT was more effective in increasing time to recurrence in patients with fewer than 12 previous episodes than in those with more than 12 episodes. The authors of the paper suggest that CBT is not as useful for those at a late stage of the illness. However considering that interepisode length often shortens as duration of illness lengthens (Goodwin & Jamison, 1990), this finding may simply reflect the greater chronicity of bipolar disorder over time (Basco et al., 2007).

New therapies with amendments to the traditional cognitive model are emerging. Ball et al (2006) reported on a randomised controlled trial of cognitive therapy with emotive schema focussed elements drawn from Gestalt therapy. The therapy is felt to be more suitable for more rigidly held beliefs, self-defeating behaviours and avoidant coping styles. In all 52 patients received either cognitive therapy or treatment as usual, the latter consisting of usual GP or psychiatrist appointments. Results showed that the CT group had a greater time to depressive relapse, although this difference was not significant at 12 month follow up when depression at baseline was controlled for. Depressive symptoms decreased significantly at baseline but not at 12 month follow up. Manic symptoms decreased over time but not significantly. It cannot be established from this design whether the emotive techniques were of additive value. Compared with promising post-

treatment results, the follow-up results suggest that benefits gradually diminished once cognitive therapy was withdrawn. The authors suggest a need for booster sessions to maintain the beneficial effects of therapy.

Vasile et al. (2007) were particularly interested in examining CBT used as an augmentation strategy for bipolar depression. In a small trial 18 bipolar patients who were currently experiencing a depressive episode serious enough to warrant admission were randomly allocated into groups to receive CBT+ drug treatment or drug treatment alone. Over 5 month follow-up the CBT group performed significantly better on various measures including depression scores and global functioning. Also the onset of antidepressant action was observed earlier in the CBT group (10.5 days compared to 17.5). The findings suggest that CBT is an effective adjunctive treatment when administered during the depressive phase and not just during periods of euthymia, although it is unclear which particular CBT strategies were employed.

Much like the psychoeducative studies, one difficulty in interpreting the results of these trials is in the relative lack of external validity. Many of the studies are comparative assessments of treatments in homogenous samples of patients, thus are not broadly representative of clinical samples. Comorbid disorders are prevalent among individuals with bipolar disorder, however rarely do studies examine the efficacy of psychological treatments for dually diagnosed patients. In fact, comorbid disorders are often an exclusion criterion for patients entering studies as they are thought to confound treatment effects. However research has begun to emerge which takes into account the complexity of this clinical population.

Group CBT has been found to be useful for individuals with concurrent bipolar disorder and substance dependence. Weiss et al. (2000) found in an open trial with Bipolar I and II that those attending an "Integrated Group Therapy" were more likely to stay abstinent from drugs and alcohol longer than those who did not attend. This is important as those individuals with concurrent substance abuse have a poor prognosis. Past studies show that in this group lithium response is poorer, suicide attempts are increased, length to stabilization during hospital stay is longer, need for inpatients admission is increased, relapse is higher, relapses are more severe, and non-compliance of medication is high (Schmitz et al. 2006). The participants of the group decreased significantly in manic symptoms and severity of alcohol-related problems, but these differences were not maintained when age was controlled for. 12-20 weekly group sessions were offered. Actual drug and alcohol use decreased but did not reach significance, suggesting a more intensive therapy is required for this group. It was also unclear whether the patients fared better because of integrated group therapy-specific content or whether they benefited from sharing their experiences in a group format.

A similar group of researchers (Weiss et al., 2007) followed this study up with a randomised trial of integrated group therapy versus group drug counselling for patients with bipolar disorder and substance dependence. Integrated Group Therapy employs a cognitive behavioural relapse prevention model which simultaneously addresses the two disorders, whilst group drug counselling focuses on substance use alone. The study compared 20 weeks of integrated group therapy or group drug counselling with 3 months post-treatment follow-up. Analysis revealed significantly fewer days of substance use for integrated group therapy patients during treatment and follow-up. No significant differences were found between the groups in the number of weeks ill with bipolar disorder during treatment and follow-up. Interestingly, integrated group therapy patients

had more depressive and manic symptoms than group drug counselling patients, a somewhat paradoxical finding given that the major difference between the groups was the integrated group therapy focus on mood. Several explanations have been offered to account for this finding. It is suggested that the differences may reflect an increased level of awareness of sub-syndromal mood symptoms among the integrated group therapy participants rather than true changes in mood. It seems plausible that the group therapy patients may have been able to identify and admit to these symptoms more readily since part of the therapy stresses the importance of early recognition of mood symptoms. An alternative explanation is that a reduction in substance abuse may worsen mood symptoms in the short-term.

An outcome study carried out by Schmitz et al. (2002) evaluated the efficacy of CBT in conjunction with pharmacotherapy for patients dually diagnosed with bipolar disorder and substance use disorder. 46 outpatients were randomly assigned into 2 groups to receive either medication monitoring (MM) and individual CBT or MM alone. The medication monitoring was designed to resemble standard outpatient clinical practice for the maintenance phase of bipolar disorder. It consisted of 4 clinic visits over 12 weeks to adjust drug doses, refill prescriptions, and discuss compliance, side effects, drug use, and mood symptoms. CBT consisted of 16 1-hour sessions aimed at reducing the probability of relapse into a manic or depressive episode, substance abuse, or both by increasingly the patient's self-awareness and ability to cope with high-risk situations. Almost double the percentage of patients in the MM + CBT group completed treatment compared to the MM group, with significantly higher session attendance in the MM + CBT group. Although the 2 groups did not differ on substance use outcomes during treatment, improvements were noted in the MM + CBT group with regards to medication compliance and mood symptoms. However the compliance improvement did not reach

significance and the mood improvements were not significant at every time period. The authors blame high attrition rates for reducing the power to evaluate post-treatment functioning.

Based on the encouraging results of studies into CBT for bipolar adults, Danielson et al. (2004) formulated a model for CBT for adolescents with bipolar disorder, intended to be used in conjunction with pharmacological treatment. Drawing on previous treatment manuals, each weekly session followed the same structure including symptom and homework review, agenda setting, teaching of new skills, and assigning new homework. Over the course of 12 weeks the treatment also included aspects of psychoeducation and family therapy. This intervention was later tested in a pilot study of 16 adolescents (aged 10-17) with bipolar disorder, with 8 enrolled in the CBT program and the other 8 in a control group (Feeny et al., 2006). Results supported the feasibility of additional psychosocial intervention. However sample size was too small to detect statistical differences in efficacy.

3.2.3 Interpersonal approaches

Inter-personal and Social Rhythm therapy (IPSRT) was yet again developed as an adjunct to long-term maintenance pharmacology. IPSRT adds a component targeting regularity in social rhythm to interpersonal psychotherapy for unipolar depression. The basis for this intervention was taken from Interpersonal Therapy for unipolar depression (Klerman et al., 1984) and from the proposals of Goodwin & Jamison (1990) that suggested 3 routes to acute episodes: i) medication non-compliance; ii) social rhythm disruption; iii) stressful life events (Frank et al., 1997). There was also consideration of the role of social and environmental 'Zeitgebers' in the development of episodes (Frank et al., 1999).

Zeitgebers were proposed as persons, social demands or tasks that set the biological clocks, and along with 'zeitstörers' (time disturbers – either physical, chemical or psychosocial events) are proposed as having an influence on the course of the disorder (Ehlers et al., 1993). One example of this would be of sleep loss and increased exposure to light, which can trigger manic and hypomanic episodes (Wehr, 1989). The therapy teaches patients about the relationship between mood and life events, while trying to help individuals overcome denial and accept the lifelong recurring nature of the disorder. It encourages individuals to develop stability in their daily routines. Monk et al. (1994) found, in a sample of the public, that lifestyle regularity was associated with fewer sleep problems and a stable circadian rhythm. Therapy should promote an understanding that life stress and the environment impact upon the onset of mood disorder symptoms, and that mood disorder symptoms in turn impact upon life stress and the environment. IPSRT aims to develop ways of maintaining standardised daily routines, particularly sleep/wake cycles when stressful events arise. At the same time it also addresses sources of interpersonal stress, in the same way as interpersonal therapy would do. Wulsin et al. (1983) described the difficulty that individuals often have in maintaining relationships and in negotiating conflict.

IPSRT combines interpersonal and environmental factors, thus covering a number of psychosocial risk areas. IPT has been found to be effective in lengthening the time between episodes (Frank et al., 1990) and in reducing relapse in the maintenance of recurrent depression (Frank et al., 1991). In a randomised controlled trial in individuals with dysthymia comparing the anti-depressant Sertraline, IPT and the Sertraline and IPT combined found that the Sertraline alone and combined groups showed more improvement in depressive symptoms than the IPT alone group. (Browne et al., 2002).

In a randomized controlled trial of IPSRT (Frank et al., 1997) 38 acute Bipolar I patients participated. In total 18 participants received IPSRT intervention and a standard Clinical Status and Symptom Review Therapy (CSSRT), and 20 received CSSRT alone. CSSRT incorporated intervention aimed at treatment adherence including education on mechanisms of action, side effects and importance of regularity. No specific methods of self-management are discussed in the paper. Over the 52 weeks of treatment all patients completed the social rhythm metric. Scores from the metric were analysed. Individuals receiving CSSRT showed no significant changes in the stability of their social rhythms implying that simply monitoring social rhythms is not sufficient. Those receiving IPSRT however showed increases in their social rhythm stability. No differences were found between the two groups in symptomatology. So although impacts were made upon social rhythm stability, these changes did not impact significantly upon symptom levels.

A randomized controlled trial of IPSRT as a maintenance therapy was conducted (Frank et al., 1997, 1999). Patients either received IPSRT or intensive clinical management (ICM). Initially patients were enrolled in one of the two therapies while they were experiencing an acute affective episode. When individuals were stable they were reassigned to either IPSRT or ICM preventative therapy. Medication was provided, initially in the acute phase a mood stabilizer was started, and other medications added as necessary. When the patient was stabilized if possible all medications bar the mood stabilizer were withdrawn. Preventative treatment included bi-weekly sessions over a period of 12 weeks, then monthly sessions for 2 years. Due to the dual randomization process it was possible that patients could either maintain the same therapy they had in the acute phase or switch. They found that participants who experienced switches in their treatment were significantly more likely to experience a recurrence than those who maintained the same treatment. There was no measure of outcome other than symptomatology and

recurrence. Members of the IPSRT group recovered more quickly from a depressive episode relative to those receiving crisis management, however there were no differences in relapse rates between the groups staying with the same therapy in both phases, i.e. those that received either ICM for both phases, or IPSRT for both phases. Increases in stability of routine were used as explanations for outcome; however there was no measurement of this.

Rucci et al. (2002) carried out a similarly designed 2-phase study examining the efficacy of pharmacotherapy with adjunctive IPSRT. Again pharmacotherapy was guided by an algorithm with the goal of reducing the use of all drugs other than lithium in the maintenance phase. The main outcome measure in this study was rate of suicide attempts. Overall 175 patients with bipolar disorder entered the study during an acute episode. Participants were treated acutely with a combination of pharmacotherapy and one of two levels of psychotherapy: IPSRT, which included help in regularising daily routines; or ICM, involving regular visits with empathic clinicians. Like the above study, participants were treated in the acute phase until stabilised, then were randomly reassigned so they could maintain the same therapy they had in the acute phase or switch. Data on prior suicide attempts were obtained retrospectively from interviews. Patients experienced significant reductions in the rate of suicide attempts during both the acute and maintenance treatment phases compared with the pre-treatment phase (3- and 17.5-fold reductions respectively). Of particular clinical relevance, no patient with a prior history of suicide attempt made a suicide attempt during the trial, although 5 other patients did make an attempt. Of these 5 patients, 4 were receiving ICM in the acute phase and 1 was receiving IPSRT in the maintenance phase. These figures point towards a proposal that IPSRT may have effects on suicidal behaviour over and above those of ICM.

However the small number of suicide attempters yielded low power for analysis and there were no significant mode-specific differences in the rates of suicide.

A more recently published account of a 2-year preventative maintenance phase of IPSRT (Frank et al., 2005) followed on from their previous randomized controlled trial of IPSRT for acute episodes. In total 175 patients received either IPSRT or Intensive Clinical Management (ICM). They were randomized into four possible treatment strategies. Patients either began IPSRT in the acute stage of their illness or ICM. Again they then continued with the same treatment into the maintenance stage or changed to the alternative treatment strategy. Participants were seen weekly in the acute phase until stabilized. In the preventative maintenance phase patients were seen every other week for 12 weeks, then monthly until the end of the 2 years. They found those patients who received IPSRT in the acute phase of their illness had a significantly longer survival time; being more likely to remain well for the two years of the preventative maintenance phase. This positive effect of acute IPSRT appears to have been mediated by the stability achieved through increased regularity of social routines. There was no significant effect of treatment type in the maintenance phase. Interestingly the study also found there was a negative effect of co-morbid medical conditions on time to remission, well time following remission, and levels of inter-episode sub-syndromal symptoms. Frank and his colleagues also found that patients with co-morbid medical conditions receiving ICM benefited more than if they received IPSRT, possibly due to the increased focus on somatic problems.

A number of factors may influence the success of treatment. One mediator in treatment success is quality and specificity of delivery. The more specific the IPT in recurrent depression, the greater the time to relapse (Frank et al., 1991). Research is required to investigate the effects of quality and specificity of IPT for bipolar disorder.

A recurring criticism of trials of all the types of psychological interventions examined in this review has been the lack of varied samples including e.g. comorbidities. Again for IPSRT it is only recently that studies have begun to include more clinically relevant subgroups. Swartz et al. (2005) recognised the unique challenges posed by patients with bipolar disorder and comorbidities. They explored the potential benefit of IPSRT in patients with bipolar I disorder and co-morbid borderline personality disorder (BPD). Interestingly, this study enrolled patients who were currently in an affective episode rather than a period of euthymia. The main outcome measure was time to stabilisation. IPSRT was specifically chosen above other types of intervention because it incorporates strategies specific to the management of acute affective episodes. In addition to this it is designed to address many of the symptoms intrinsic to both bipolar disorder and BPD.

Overall 58 patients with bipolar disorder and 12 patients with bipolar disorder and BPD received IPSRT and medication. Only 25% of the bipolar+BPD patients achieved stabilisation compared with 74% of the bipolar-only patients. Those in the bipolar+BPD group that did stabilise took 34, 96, and 97 weeks respectively to stabilise, whilst those in the bipolar-only group took an estimate median time of 34.7 weeks. Both groups improved in symptom severity over time. It is difficult to know what conclusions to draw from the results of the study as many potential confounding factors were at play. The dropout rate in the comorbid group was high for various reasons. The patients were all experiencing at least their third affective episode making this a fairly chronic group. The bipolar+BPD group had more depressive symptoms at baseline which may have contributed to longer time to remission. This lack of a control group e.g. bipolar+BPD patients receiving pharmacotherapy alone, makes the effect of IPSRT difficult to evaluate. However, the fact that despite not fulfilling criteria for remission many bipolar+BPD

patients remained in treatment for a long period of time and improved significantly in symptomology, the benefit of IPSRT is perhaps indicated. The authors suggest that a hybrid psychotherapy designed specifically to address the needs of this population and their unique characteristics may lead to better outcomes.

3.2.4 Family Therapy approaches

The development of family therapy like the other therapies was motivated initially to aid medication compliance (Miklowitz & Goldstein, 1990). However it has also been influenced by the research into Expressed Emotion (EE) in family environments. EE is thought to consist of critical comments from relatives, hostility and emotional over-involvement (Brown et al, 1958). EE has been highlighted as a psychosocial risk factor in the development of Bipolar Disorder, and more broadly as a general predictor of outcome in a number of psychiatric disorders, especially schizophrenia, where strong relations between EE and outcome have been found (Butzlaff & Hooley, 1998). Research into family therapy for schizophrenia has had good results, with decreases in hospitalisation time, lower levels of family burden (Xiong et al., 1994), increases in functioning and improved family attitudes (Glick et al., 1985; Haas et al., 1988), decreases in expressed emotion, symptomatology and in relapse rates (Leff et al., 1982; Falloon et al., 1982; Hogarty et al., 1986; Tarrier et al., 1988; Leff et al., 1989).

As discussed in more detail in Chapter 2 above, in bipolar Disorder higher rate of relapse has been associated with higher levels of EE within a family, particularly in the post discharge period (Miklowitz et al., 1988). The direction of causality is difficult to ascertain and relapse could indeed be predictive of high EE rather than vice versa. (Reinares et al., 2002). Honig et al. (1995, 1997) found that levels of EE decreased, although not

significantly, in individuals taking part in a multi-family psychoeducational intervention. Low EE was in turn associated with a lower rate of relapse and previous admissions, suggesting that reducing high expressed emotion is an important goal of any family intervention.

Family therapy is holistic and focuses on maximising positive communications within the family, functioning within the family and coping following a bipolar episode. As a consequence most family therapy treatments are psychoeducational in form; they teach family members about the nature of the disorder, available treatments, and acceptance of relapse, effective communication and problem solving (Miklowitz & Hooley, 1998). An understanding of the extent of the burden that is placed on the families of individuals with Bipolar Disorder is necessary. This burden is present in a number of domains including social, leisure, and financial. Therapy not only aims to reduce relapse in the patients but to be protective of the mental health of the family members (Reinares et al., 2002).

The relative success of Family Focused Therapy (FFT) with treatment as usual has been the subject of research. Miklowitz & Goldstein (1990) first developed their family treatment package from the model developed by Falloon and colleagues (1984) with schizophrenia patients. It is a home-based psychosocial treatment built of psychoeducation, communication skills and problem-solving skills. In Miklowitz & Goldstein's (1990) study there were 21 one-hour sessions over 9 months in a home-based setting. Outcome was measured on the basis of relapse. They found that 11% of the treatment group relapsed over the 9 month period compared to 61% in the control group. The study was limited by its small sample size and unequal group sizes, making it difficult to generalise the findings. Family therapy was more effective when it was more flexible, less didactic and directed at higher functioning individuals.

Clarkin and colleagues (1990) reported in a series of papers on a randomized controlled trial of 'Inpatient Family Therapy' in addition to standard hospital treatment. (Haas et al, 1988; Spencer et al; 1988; Clarkin et al, 1990; Glick et al.,1991, 1993). Therapy was designed to improve family attitudes towards the patient and towards their treatment, thereby increasing treatment compliance. The study included 50 individuals who had a variety of diagnoses under the general umbrella of major affective disorder. The treatment group were compared with a group who received standard hospital treatment only. Interestingly, at 6 months and 18 months after therapy, bipolar patients showed a better outcome while unipolar individuals did better without it. It was hypothesized that this finding was due to some similarities between bipolar illness and schizophrenia, the latter of which the therapy was originally designed for. Analysis of mediating factors found that psychosocial treatment compliance and reduction in family rejection of the patient were both correlated with good outcome (Glick et al., 1991). The study must be interpreted with caution, as this was a relatively small sample, with only 21 participants having bipolar disorder.

Simoneau et al. (1999) compared a family-focused psychoeducational therapy with crisis management with a naturalistic follow-up both with maintenance pharmacotherapy. They specifically examined the effects of the intervention on family communication and problem solving. FFT increased positive non-verbal interactions compared to the crisis management group, but did not have a corresponding decrease in negative interactional behaviours. Verbal behaviour was not affected. Symptomology improved in the FFT group in comparison to the crisis management, an effect found to be mediated by the increase in positive nonverbal behaviours. A later small randomised controlled trial of psychoeducation with relatives (Reinher et al, 2004) found the treatment reduced the

burden of the caregiver. Similarly, a trial of cognitive-psychoeducational therapy with bipolar patients and their relatives reduced objective burden, high expressed emotion and depressive symptoms of the relatives at 1 year follow up (Bernhard et al., 2006). However, there have also been negative results. A trial of psychoeducational multifamily group therapy in Bipolar I patients found no difference in time to recovery (Miller et al., 2004). However, this study did not consider other outcomes such as functioning and relapse.

Mikowitz and his colleagues reported on relapse rates in the same study (Miklowitz et al., 2000) using the same 9 month Family focused treatment paradigm designed by Miklowitz & Goldstein (1990). At 2-year follow up they found 1-year survival rates (without relapsing) of 71% in the Family focused treatment group and 47% for the treatment as usual group. Family focused therapy was found to significantly reduce the time to relapse. This improvement was maintained when severity and polarity of acute episode at entry, inpatient status, the number of lifetime episodes and various demographic variables were controlled for. There was a significant improvement in symptoms in the treatment group, with individuals in depressive phases benefiting most from the intervention. However when EE was used as a covariate there was still a main effect of treatment but there was no main effect of EE and there was no significant interaction. The study was limited as there was no control for the increased contact the treatment group had with services. The study also required that the group had to be compliant with medication and this compliant nature may have meant that the group was not truly representative of the bipolar population. The above study was strengthened by its consideration of long term outcomes. Outcomes over a further one year period were looked at for the above study (Miklowitz et al., 2003a). Overall results suggested that

manic and depressive symptoms may be influenced by different constellations of risk and protective factors.

Interventions specifically for spouses have also been developed. Van Gent & Zwart (1991) compared the outcome of five sessions of psychoeducation in partners of 14 bipolar patients with 12 partner controls who did not attend therapy. The aims of psychotherapy were stated as increasing partner's knowledge of the disorder, medication and social strategies. Knowledge was found to increase, however measures of interactional problem solving, psychosocial problems and patient's compliance did not change.

Similarly, Clarkin and colleagues (1998) allocated 19 patients to receive medication and marital therapy and 23 received medication alone. The marital therapy was carried out over 11 months and consisted of 25 sessions. The content of the sessions was psychoeducational in nature. The authors found those who received the therapy showed greater improvement in overall functioning and greater medication adherence; therapy did not improve symptom levels over and above the improvements due to the medication. However the sample was small, consisting of a narrow group of bipolar patients in their middle age who had been married for on average 17 years. The sample may not have been representative of the bipolar population and may not have been likely to benefit significantly from the intervention.

A pertinent question is whether family therapy, with its associated difficulties in organisation and compliance, is superior to individual therapy? Rea et al. (2003) carried out a study comparing family-focused and individual therapy. On the whole 53 families participated with 28 in the family-focused treatment and 25 in the individual treatment. The therapy was conducted for 21 sessions over a 9-month period, and a follow-up was

conducted 1 year post treatment. They found 28% of patients in the Family-focused treatment relapsed in the post-treatment year compared to 60% in the individual treatment. They also found only 12 % of patients in the family-focused treatment were rehospitalised compared to 60% in the individual treatment. The authors suggest a protective effect of family therapy against hospitalisation at times of relapse. A further finding during treatment suggested that Family Therapy protected individuals with poor premorbid adjustment from relapse. Such protection was not found with individual treatment. Findings from the study are supportive of family-focused therapy as an effective psychosocial treatment for outpatient care. As with previous studies there was no adequate control for increased contact and it did not include medication refusers. Also in future studies it would be relevant to examine the levels of expressed emotion in the families. It may be that family therapy is better for high EE families only. This is certainly an area worthy of future investigation.

Few treatments have focused on the therapeutic needs of both the patient and family needs. Aiming to reduce family burden and reduce expressed emotion in the family, Bernhard et al. (2006) established a hospital based cognitive-psychoeducational program. Sixty-two patients attended 14 sessions of group therapy, and 49 relatives received 2 psychoeducational workshops of 4 hours each. Most of the patients began the group after an acute episode. There were significant improvements in patients' knowledge of bipolar disorder, a significant reduction in symptom related burden after the intervention and at 1 year follow-up, and in expressed emotion and objective burden at 1 year follow-up. The results are limited by the lack of control group. As the authors state, minor symptoms still present may have affected relatives' burden and attitude toward the patient.

A recent development has been the integration of individual family therapy with interpersonal and social rhythm therapy; both therapies target important risk factors in the development of bipolar disorder including EE, stressful life events and social rhythm disruptions (Miklowitz et al., 2003b). One open-trial has been carried with patients following an acute episode. It included up to 50 weeks of individual and family therapy combined with mood stabilising medication. Comparisons were made with a group receiving a standard community care package (2 family education sessions, mood-stabilising medication and crisis management) from a previous study. They found individuals receiving the integrated therapy had a longer time until relapse and greater reduction in depressive symptoms over a 1 year period than those receiving standard care. However the actual quantitative difference in relapse (30% in IFIT and 39% in CM) was non-significant. It would be useful for the results of this study to be replicated with a randomised controlled trial where amount of contact with services are controlled for.

Research into family therapy for schizophrenia has found that multi-family groups are superior and more cost effective to individual family therapy in reducing relapse (McFarlane, 1990, 1994). However in bipolar disorder the only study in the literature to compare multi-family group therapy with individual family unit therapy (Miller et al., 2004) found no differences between the groups in the proportion of patients who recovered or in time to recovery. Nor did these two groups differ from the control group of psychotherapy alone. It seems the outcome measures were insufficient to detect a difference between the groups but perhaps additional measures such as psychosocial functioning or quality of life may have drawn some more positive findings. Further research is needed to compare the outcome of individuals attending multi-family groups versus individual family unit therapy.

Similar to the other therapies, it is once again the case that studies examining the role of FFT in more diverse groups of bipolar patients are uncommon. However, in FFT there is a body of research pertaining to children and adolescents with bipolar disorder; more so than for other types of intervention. Perhaps this is because family therapy seems a logical and appropriate treatment for these groups given that they typically live with relatives who are responsible for their care.

Most of the work on FFT for childhood bipolar illness has been carried out by Fristad and colleagues. Based on previous studies with adults, Fristad et al. (1996) developed adaptations to family psychoeducation to make it developmentally appropriate for families of children and adolescents with mood disorders. This program was further developed and tested by Fristad et al. (1998). Pilot data from families of 3 children and 6 adolescents indicated a strong endorsement of the program. Family members reported increased understanding of the disorder and drug treatments, increased awareness of family interactions, and believed the sessions covered appropriate material well. It should be noted that this small study included children and adolescents with all types of mood disorder so the results are not related to bipolar disorder alone.

Fristad et al. (2002) followed this pilot study up with a randomised controlled trial involving 35 children aged 8-11 years and their parents. Families were randomized into immediate Multifamily Psychoeducation Groups (MFPG) plus treatment as usual (TAU) or wait-list plus TAU. Both groups contained children with bipolar disorder and major depressive disorder. The group format allowed parents to meet other parents dealing with the unique pressure of bringing up a child with a mood disorder and gain support from each other. Clinical impressions suggested that combining families of children with bipolar and depressive illnesses is feasible and potentially beneficial. Immediately and 4

month-post treatment, both sets of families described having gained knowledge, skills, support, and positive attitudes during treatment. Miklowitz et al. (2004) have also developed a family focused treatment for adolescents with bipolar disorder. An open trial with 20 bipolar adolescents found that FFT developed for adolescents and pharmacotherapy was associated with improvements in depressive symptoms, mania symptoms, and behavioural problems over 1 year.

Aside from the work carried out with children and adolescents, studies examining the role of FFT in other subgroups of bipolar patients are rare. Miklowitz & Taylor (2006) present a review of FFT in the treatment of suicidal bipolar patients. They describe how the 3 elements of FFT (psychoeducation, communication skills training, and problem solving) can be adapted to address the specific needs of suicidal bipolar patients. The main goals of the intervention are to help the family to understand that suicidal feelings and behaviours are part of the pathophysiology of the disorder but are also under environmental control to a certain extent. The family are encouraged to become involved in the development and implementation of a suicide prevention contract in which environmental triggers are identified and what each person should do if a trigger arises. They present a single case study to illustrate the application of their modified FFT but, to date, no trials address this subgroup directly.

One of the particular benefits of FFT is that it can begin early on in the treatment of a patient with bipolar disorder and can be continued during acute phases. During this time the severity of the patient's symptoms may mean they are not stable enough to tolerate much therapy, but much psychotherapeutic work can be done with the family (Basco et al., 2007).

3.2.5 Long term and psychiatric management issues

Although an attempt has been made in this review to examine the different types of psychological interventions separately, many of the studies reviewed above integrate more than one component in their approach. Psychoeducation in particular seems to play a role in most interventions, whether the authors acknowledge it or not. Even in studies of interventions clearly stated as psychoeducational, treatments delivered with a large number of sessions probably incorporate other therapeutic elements (Rouget & Aubry, 2007). This makes it particularly difficult to tease apart what is actually causing the therapeutic effects. How do we know which processes are contributing and indeed what their contribution really is? Rouget & Aubry (2007) concede that this issue cannot really be resolved given the overlap of the various interventions. Their review is the only one in the literature to examine the efficacy of an intervention (psychoeducation) by distinguishing between different therapeutic targets, rather than classifying studies by the type of approach.

Recent studies have introduced a multicomponent care program incorporating elements adapted from several types of psychological intervention. Simon et al. (2002; 2005; 2006) evaluated a multi-program care management program in a large sample with minimal exclusion criteria. The program included five core elements of care planning, monthly telephone monitoring, feedback, a structured group psychoeducational program, and support, education and care coordination, all delivered by a nurse care manager. The assessment and care planning involved the nurse care manager developing a collaborative treatment plan including early warning signs of mood episodes and coping strategies for responding to these signs. The telephone monitoring involved monthly calls to complete ratings scales and support patients' self-monitoring. Following each call feedback was then

given to the usual treating mental health providers (psychiatrists and psychotherapists). The group psychoeducational program was adapted from Bauer and McBride's Life Goals Program (Bauer & McBride, 2003) which included the creation of a self-management plan with prodromes and coping strategies. Phase 2 of the Program involved a structured problem-solving format to focus on accomplishment of specific life goals. The treatment group was compared with treatment as usual. The intervention group had significantly lower mania scores in the 12- (2005) and 24- (2006) month follow-up periods and spent significantly less time in hypomanic or manic episodes. Depression declined over time for both groups but the two groups did not differ significantly in depression scores across the follow-up period. These polarity findings replicated those found by Perry et al. (1999). The authors suggest that the incorporation of more specific cognitive and behavioural content may be necessary to achieve improvements in depressive symptoms. This randomised trial differed from others in the inclusion of patients with a range of baseline symptoms, thus providing a more realistic idea of effectiveness in the clinical population. However the benefits of the intervention program were limited to patients with significant baseline mood symptoms suggesting that this level of input has limited benefit to those who fare well with usual care.

Bauer et al. (2006a,b) identified a need for an intervention to (a) accommodate severely ill patients with comorbidities, typically excluded from trials, (b) minimise variability of care provided, and (c) minimise system-related barriers, allowing providers and patients to come together for illness management. They created a care model based on the lithium clinics of the 1970s and enhanced by principles of chronic care models for medical illnesses. The intervention was team-based and consisted of three components to address patient, provider, and system aspects of care. To improve self-management skills participants were enrolled in psychoeducation, with focus on personal symptom profiles,

early warning symptoms, and triggers. Simplified clinical practice guidelines were provided to offer guidance to providers for drug treatment decision making and were regularly updated. Finally a nurse care coordinator was used alongside the psychiatrist to improve continuity of care and information flow.

An early pilot study (Bauer et al., 1997) showed promising results, with patients reporting greater treatment satisfaction compared to baseline measures, high adherence to the intervention program after one year, and reduced days spent in psychiatric hospital compared to the year prior to treatment. In total 330 participants were randomly allocated to the intervention group or treatment as usual in the larger study (2006a,b) and outcome data was collected for 306. Follow-up over three years was completed by 80% of participants, with no difference between intervention and treatment as usual groups. Results showed that the intervention significantly reduced weeks in affective episode, primarily mania, consistent with the findings of the other multicomponent care program (Simon et al., 2006). Broad-based improvements were demonstrated in overall social function, mental quality of life, and treatment satisfaction, despite no significant reductions in depressive symptoms. Two possible explanations are offered for this finding. The first is that reductions in mania and modest reductions in depression were sufficient for significant functional gains. However this is unlikely as studies indicate that ongoing depression is the strongest correlate of functional deficits in bipolar disorder (Bauer et al., 2001). The second explanation proposed is that the combination of psychoeducation and facilitated collaboration with providers may have helped participants to manage their lives more effectively despite ongoing depressive symptoms. The authors purport that multiple components contributed to the findings. Treatment satisfaction was also higher in the intervention group from six-months onwards. Interestingly, no

significant positive effects on symptoms or general functioning were evident at the end of year one, highlighting the long-term rather than short-term benefits of therapy.

Continuing the trend for multicomponent care packages, the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD, Sachs et al., 2003) is a unique public health initiative designed to examine the effects of various treatments and their impact on bipolar disorder in the community. It represents the largest treatment study of bipolar disorder ever performed. The overall program combines a large prospective naturalistic study and a series of RCTs that share a battery of common assessments. It explores the effects of psychosocial interventions on community samples of patients followed naturalistically, thus meeting the challenge of being an effectiveness study in a broadly representative sample. As well as ensuring wide heterogeneity of the bipolar spectrum (with virtually no exclusion criteria for the overall program), this program was designed to try and bridge the gap that can occur between RCTs and the treatment of patients in clinical practice. The overall study design allows the results from methods that enhance internal validity to be compared with those that enhance external validity (Sachs et al., 2003).

All participants in the STEP-BD Program receive standard care pathway (SCP) treatment. In addition, participants may also receive randomised care pathway (RCP) treatment with one of three psychotherapies: CBT; FFT; or IPSRT. Randomised patients complete 30 sessions over 9 months in the care package, then return to SCP. Patients can only enter each randomised intervention once, but remain eligible for other RCPs after they return to standard care.

As well as measuring treatment effectiveness, the program has the objectives of estimating costs and quality of life outcomes of both acute treatment and long-term management strategies. The study also aims to assess the prognostic impact of comorbid conditions and examine various complexities within the illness itself. The program has so far yielded numerous publications with promising results which have been examined throughout this review (e.g. Perlis et al., 2004, 2006). In particular, as noted by DePaulo (2006), the outcomes reported in the Perlis et al. (2006) paper are better than those reported by similar scale effectiveness studies of schizophrenia (CATIE, Lieberman et al., 2005) and major depression (STAR*D, Trivedi et al., 2006).

The implementation of these large multicomponent treatment programs is evidence of a shift in recent research from efficacy programs to effectiveness research in the bipolar population. The consistency of findings between the studies led by Simon and Bauer, conducted concurrently among different populations and systems, supports the feasibility and effectiveness of such care models. Most importantly a shift has also occurred from simply measuring outcome in terms of symptoms and medication adherence, to more viable outcome measures such as functional recovery. However because of the various psychological elements making up the intervention package it may be difficult to tease apart the particular cause of improvement. One of the limitations of different interventions being used together meant one cannot separate the respective efficacy of each (Gonzales-Pinto et al., 2004).

3.3 Summary

There is consistent evidence in the literature supporting the role of psychoeducation, cognitive-behavioural therapy, family-focussed therapy and IPSRT in the treatment of bipolar disorder. They have targeted relapse, prevented hospitalisation, reduced

symptoms, and improved functioning. However, authors have pointed to the variability in outcomes as pointing to a need for further quality large-scale randomised controlled trials (Gutierrez & Scott, 2004). It is not yet possible to say conclusively which type of episodes each therapy best targets, and at what stage of the illness is the therapy best used. However, CBT trials overall have been more successful at targeting depressive episodes, and IPSRT/FFT at manic episodes. Miklowitz and Craighead (2001) suggest the combination of these therapies would therefore make sense. Given the mounting evidence that shows that residual symptoms are significantly associated with shorter time to relapse, targeting subsyndromal symptoms in maintenance treatment may also represent an opportunity to reduce risk of recurrence (Perlis et al., 2006; Paykel et al., 2006).

In general there has been an over reliance upon symptom measures to determine efficacy without considering the psychosocial and psychological theoretical concepts that have influenced the development of the therapy. Also neglected are changes in general functioning outcomes and quality of life.

Numerous studies have reported deficits in quality of life among individuals with bipolar disorder (e.g. Cooke et al., 1996; Robb et al., 1997; Leidy et al., 1998; Arnold et al., 2000; NacQueens et al., 2000; Vojta et al., 2001; Yatham et al., 2004; Sierra et al., 2005). Quality of life encompasses those aspects of life that make it particularly fulfilling and worthwhile (Akvardar et al., 2006). These extend beyond traditional symptoms and include subjective feelings of wellbeing, satisfaction, functioning and impairment (Quilty et al., 2002). Bipolar disorder can adversely affect most aspects of life, particularly physical and psychological aspects as well as social, occupational, and economic status. Although poor quality of life has been shown to have a strong relationship with depression (Vojta

et al., 2001; Zhang et al., 2006), the long-term effects usually remain despite remission of symptoms. Sierra et al. (2005) found that quality of life was significantly lower in a group of euthymic patients compared to controls on all 8 subscales of the SF-36 questionnaire (adapted for Spanish, Alonso et al., 1995; Original SF-36 by Ware et al., 1993). The 8 subscales encompass health concepts important to quality of life including physical functioning, role limitations due to physical health, bodily pain, general health, vitality, social functioning, role limitations due to emotional problems, and mental health.

Caryell et al. (1993) found that even when patients have been symptom free for up to 2 years, psychosocial impairment was highly apparent and not diminished by the symptom free period. Furthermore, these residual quality of life issues may in fact be sufficiently stressful to precipitate a relapse (Akvardar et al., 2006). The impact of quality of life issues should not be undervalued, and psychosocial measures may be considered as more viable outcome measures. Relapse is almost a rule but increases in ability to cope with these relapses may reduce their impact and duration, thus increasing measures of quality of life. Researchers have started to recognise that effective treatment may be more importantly measured on the basis of functional recovery and not on symptomatology (MacQueen et al., 2001; Miklowitz & Johnson, 2006).

There is a dearth of study targeting psychological interventions for prodromal individuals at high risk of developing bipolar disorder. Henin et al. (2007) found that bipolar disorder in adults is frequently preceded by childhood disruptive behaviour and anxiety disorders. Many of these childhood disorders may persist into adult life and can complicate the course of bipolar disorder in adulthood, although further investigation is required to clarify their true impact. The authors of this study suggest it may be important to identify and target childhood disorders as antecedent conditions or early manifestations of

bipolar disorder. Increased knowledge of the early expression and development of the disorder may help the development of specific preventative and early intervention strategies. Given that many at risk individuals are adolescents this makes investigation of family risk and protective factors i.e. EE, family functioning, particularly relevant to the design of appropriate early interventions. Another high risk group of people are patients who have experienced a depressive episode but have not (yet) attracted a diagnosis of bipolar disorder as they have not had an episode of (hypo)mania. The polarity of first-episode tends to be depressive (see introduction) and crucial work could potentially be done in the pre-diagnosis 'waiting' period by investigating further developmental markers and common prodromal features.

As well as the growing evidence that psychological interventions are effective for treating bipolar disorder, it seems that they are gladly received by patients too. Surveys of patient organisations in the US and the UK reveal that patients are keen to receive both self-help and psychological treatments in addition to drug treatment (Lish et al., 1994; Hill & Shepard, 1996). Psychological therapies are popular with many patients because they promote self-efficacy – the patient is doing something themselves to increase control of their problems (Morris, 2006). Patients complain bitterly about side effects of drug treatments and low compliance shows that alternatives are not just needed but are also hugely sought after. The benefits of being an appealing treatment that patients strongly desire may include increased compliance and subsequent enhanced effectiveness. On the other hand, participation rates in some intervention programs are nevertheless moderate, suggesting not all patients find this type of intervention appealing. Even et al. (2007) investigated the characteristics of patients who were willing to participate versus non-participants of a psychoeducation program. They found that the older, the less educated, those who had less knowledge about their treatment, and those with a more

external locus of control were less likely to take part. Paradoxically these are the patients who perhaps need it most. It is important to note that all patients were euthymic when approached to take part in the program so it is unlikely that mood symptoms affected their decision to take part. The groups showed no significant differences in terms of gender ratio, depression scores, duration of lithium treatment, attitude towards lithium, or time since last episode. There is scope for further research to elucidate what factors put people off from participating in intervention programs, and subsequent development of methods to encourage them.

It should be taken into consideration that many of the interventions described in the studies above involve extensive hours of therapy with highly trained therapists. This evidently places high strains on NHS resources and restricts their potential availability. Only recent studies have examined the cost-effectiveness of their interventions. Lam et al. (2005b) found there was a high probability of cognitive therapy being cost-effective for individuals with bipolar I disorder over a 30-month period. Lesser costs of treatment were due to an increased number of illness free days, and therefore a lesser load on standard psychiatric services. Bauer et al. (2006b) found their collaborative care intervention to be cost neutral with costs of intervention again being offset by reductions in inpatient costs. In a global study, Chisholm et al. (2005) measured the cost-effectiveness of clinical interventions for reducing the burden of bipolar disorder. They found that community-based treatment with Lithium and the addition of psychosocial care was most cost-effective in both developing and developed sub-regions. Furthermore this study confined the effects of psychosocial treatment to improving drug adherence so the true cost-effectiveness of psychosocial intervention was perhaps underestimated. However not all studies have investigated whether their interventions are so cost viable. Lobban et al. (2007) highlight the need for interventions to be offered in more cost-effective ways. They have devised a trial assessing the feasibility of training CMHT care

coordinators to offer enhanced relapse prevention for individuals with bipolar disorder. Results have not yet been published but the authors hope they will indicate that this is a workable approach.

Another potential way to make interventions more widely available is the use of groups in therapy. Group treatments could be a cost-effective alternative to individual therapy, which requires many hours of therapist contact. Some patients benefit from group work as it helps them to accept their disorder, interact together, and feel less stigmatised in the company of others also dealing with the illness. They may also bounce ideas off each other and acquire new ways of managing their illness through sharing their experiences. This was confirmed by patients taking part in the Life Goals Program open study (de Andrès et al., 2006) who acknowledged they were especially satisfied with the advantages stemming from group interactions. The benefits of group therapy for sharing ideas and support were also expressed in the Palmer & Williams study (1995).

Despite the benefits of group therapy, studies comparing group versus individual therapy in most of the above interventions are limited and findings largely anecdotal. This is an area of research open to future exploration, specifically, is either therapy more effective than the other? What are the respective adherence rates? Which do patients prefer?

The interventions examined in this review focus on treating the affective symptoms of bipolar disorder and the development of skills to deal with psychosocial stressors. To date, no studies examine interventions specifically for psychosis in bipolar disorder. Studies suggest that psychosis is present in up to 75% of manic patients (Tohen et al., 1990). There are studies which examine the role of psychological therapy in psychosis, but not many differentiate between psychosis in the context of bipolar disorder from

psychosis in a background of schizophrenia/schizo-affective disorder. It is debateable whether this distinction is necessary and there is much discussion in the literature, outwith the scope of this review, regarding the dichotomy versus spectrum debate. Psychological services in particular have shown a trend for treating symptoms rather than diagnoses. Numerous questions emerge, such as do patients with bipolar disorder experience different types of psychotic symptoms? For example are they more likely to experience mood congruent delusions/hallucinations? Given that patients are more likely to experience psychotic symptoms when manic, are they thus related to affect? Are there different mechanisms behind bipolar psychoses? Does this indicate a rationale for different treatment? Macmillan et al. (2007) examined the proportions of all diagnoses in people presenting to an early intervention in psychosis service in Norfolk and compared symptoms and outcome between diagnostic groups. They found that bipolar disorder accounted for around 20% of referrals, which they deemed a significant proportion. Patients with psychosis and bipolar disorder showed significantly lower rates of negative symptoms than other psychoses groups and better social functioning. Negative symptoms were found to be the only significant predictor of outcome at 1-year follow-up. They concluded that diagnosis-specific early intervention treatments may be particularly helpful to people with bipolar disorder with psychosis. The psychological treatment of psychotic symptoms in bipolar disorder is certainly an area worthy of future research.

The psychological treatments examined in this review differ in their mechanisms of action, when they are initiated, and in what format they are delivered. It appears that while most results of these studies are encouraging, further research is required to identify the specific mechanisms involved in such improvements (Scott, 2001), and to which patient groups the findings relate.

Table 3.1 offers an overview of all reviewed psychological intervention studies, summarising design and definition of outcomes. This overview aids the direct comparison of the methods, measures, and the variability of outcomes in psychological intervention trials in bipolar disorders to date.

In the next section the different psychosocial and psychological variables that have driven the development of the above therapies will be discussed in more depth. Attempts will be made to try and build a picture of the interacting variables that influence long-term outcome in bipolar disorder and the subsequent implications for effective treatments.

Table 3.1: CBT and IPSRT outcome studies

Author	Date	Title	Type of Study	Outcomes
CBT				
Cochran, S.D.	1984	Preventing medical non-compliance in the outpatient treatment of bipolar affective disorders.	RCT – standard care versus standard care plus intervention	Main outcome measure - compliance of lithium evaluated by Compliance Index Rating (combination of self-report, informant report, physician report and lithium levels) Other outcome measures - number of affective episodes and number of hospitalisations, assessed by chart review. Participants in the intervention group had better composite adherence scores as assessed by medication compliance, appointment attendance, and lithium levels, than the control group during treatment and at 6-month follow-up. The intervention group were also significantly less likely than controls to have major compliance problems, terminate lithium treatment against medical advice and to be hospitalised.
Palmer, A.G., Williams, H. & Adams, M.	1995	CBT in a Group Format for Bipolar Affective Disorder.	Exploratory study (n=4)	Outcome was measured through changes in symptom levels, NHS resources used and social functioning. No consistent change across the four participants; no generalisable results but individual reports of relative improvement in general symptoms (ISS, SCL-90) and social functioning (SAS).
Hirschfeld-Becker, D.R., Gould, R.A., Reilly-Harrington, N., et al.	1998	Short-term adjunctive cognitive-behavioural group therapy for bipolar disorder: preliminary results	RCT, psychoeducation and group CBT (n=15). Follow up at 6, 12 & 18 months.	In the CBT group significantly longer periods of euthymia and fewer new episodes, both at end of treatment and 18 month follow up.
Zaretsky, A.E., Zindel, V.S. & Gerner, M.	1999	Cognitive therapy for bipolar depression: a pilot study.	Matched-case control: bipolar patients in depressed phase versus unipolar currently depressed	Outcome measures – HDRS, DAS, and BDI. Bipolar patients achieved similar levels of reduction in depressive symptoms following CBT as unipolar patients. Significant decrease in pre- to post- scores on the BDI for both groups. Bipolar patients showed, on average, well over a 50% reduction on the HDRS, comparable to that observed in the matched unipolar patients. However on measures of more pervasive dysfunctional attitudes, bipolar patients did not improve to the same degree. Although negative automatic thinking for the bipolar group was reduced,

Lam, D.H., Bright, J., Jones, S., Hayward, P., Schuck, N., Chisholm, D. & Sham, P.	2000	Cognitive therapy for bipolar illness - a pilot study of relapse prevention.	N=13 euthymic bipolar I patients. Random allocation to CBT and TAU only. Follow up at 6 months.	<p>scores on the DAS did not decrease significantly following CBT, whereas the opposite was true for the unipolar subjects.</p> <p>Outcome measures – MAS, Internal State Scale (ISS), BHS, MRC Social Performance Schedule (SPS), Self-Control Behavior Schedule – Coping with Depression, Early Warning and Coping Interview, Medication Compliance Questionnaire (MCQ).</p> <p>Episodes – There were significantly more manic, hypomanic, depressed, total bipolar episodes, and hospitalisations in the control group than in the therapy group. After adjusting for gender, previous episodes, previous hospitalisations, and suicide attempts, the results were still significant for hypomanic episodes and total bipolar episodes.</p> <p>Other outcomes – All the mood measures (BDI, BHS, HRSD, and MAS subscales) showed a similar trend. At 6 and 12 months the therapy group had lower mean scores than the control group, although not significantly so.</p> <p>BHS – The therapy group had significantly lower hopelessness scores at 6 months but the significant difference disappeared at 12 months.</p> <p>SPS – The therapy group did significantly better at 6 months and 12 months in terms of social performance.</p> <p>SCBS – The therapy group did significantly better at 6 months and 12 months in terms of coping. The therapy group did significantly better with coping with prodromes of mania at 6 and 12 months, but only at 12 months with prodromes of depression.</p> <p>ANCOVA showed the same pattern of results with the baseline differences controlled for all measures except HRSD. With HRSD the therapy group was significantly less depressed at 12 months.</p> <p>Monthly measures – Therapy group had significantly lower mean BHS scores over the 12 months and significantly better compliance.</p> <p>BHS and MCQ – significant effect over 12 months due to significant last 6 months – difference was nonsignificant at 6 months.</p> <p>Trend for therapy group to score lower on ISS subscale all</p>
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Weiss, R.D., Griffin, M.L., Greenfield, S.F., Najavits, L.M., Wyner, D., Soto, J.A. & Hennen, J.A.	2000	Group therapy for patients with bipolar disorder and substance dependence: Results of a pilot study.	Open trial. 45 patients with bipolar disorder and substance dependence recruited in sequential blocks to receive group therapy (employing a cognitive-behavioural relapse-prevention model; n=21) or assessments but no treatment (n=24). No random assignment, but subjects could not choose either condition.	<p>through the 12 months. Difference was significant for first 6 months but not for last 6 months.</p> <p>Fluctuations – The therapy group had a significantly lower mean ISS Activation fluctuation score, a significantly lower ISS Depression fluctuation score, and a significantly lower MCQ fluctuation score.</p> <p>Medication – There were significantly less neuroleptics prescribed in the therapy group for the 6 months post-therapy.</p> <p>Outcome measures:</p> <p>Substance use – Addiction Severity Index (ASI), the Timeline followback assessment, urine toxicology screens, breath alcohol assessments.</p> <p>Mood symptoms – YMRS, HAM-D</p> <p>Medication compliance – compliance interview (adapted)</p> <p>Mood outcomes:</p> <p>Integrated group therapy (IGT) patients had a significantly greater improvement in YMRS scores than did the non-IGT patients</p> <p>No between group differences were found for change in HAM-D scores over time.</p> <p>Other outcomes:</p> <p>Substance use – IGT patients had significantly better outcomes on the ASI drug composite score, percentage of months abstinent, and likelihood of achieving 2 or 3 consecutive months abstinent.</p> <p>Compliance – Although IGT patients reported higher compliance than non-IGT patients every month, no significant difference by IGT status was found for change in compliance scores over time.</p> <p>Hospitalisation – There were no significant between group differences in the number of patients hospitalised during the study period, nor the mean length of time per hospitalisation. One non-IGT patient committed suicide during the study period.</p> <p>Relevant:</p> <p>Because subjects were not randomly assigned to treatment groups, it is possible that those with clinical or sociodemographic characteristics associated with good outcomes were more likely to enter IGT. Comparison of IGT and non-IGT subjects showed that age was nonrandomly distributed between the two groups. Thus, treatment condition and age were entered together in multivariate</p>
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Fava, G.A., Bartolucci, G., Rafanelli, C. & Mangelli, L.	2001	Cognitive-Behavioural management of Patients with Bipolar Disorder who relapsed while on lithium prophylaxis	Naturalistic open clinical trial. 15 patients who relapsed while on lithium were treated with CBT. 2 to 9 year follow-up was performed.	<p>regression analyses with the outcome measures in which there were significant treatment group differences. For change in ASI alcohol composite score and YMRS score, neither significant treatment group difference was sustained after age was added to the model.</p> <p>Outcome measures – survival analysis – time to relapse and BPRS. Follow-up evaluations ranged from 24 to 108 months. CBT was associated with a significant reduction in residual symptomatology as measured by BPRS scores. While all patients had a relapse within 30 months of starting lithium (pre-CBT treatment period), only 27% relapsed in the same time period following CBT treatment.</p>
Patelis-Siotis, I., Young, L.T., Robb, J.C., Marriott, M., Bieling, P.J., Cox, L.C. & Joffe, R.T.	2001	Group cognitive behavioural therapy for bipolar disorder: a feasibility and effectiveness study.	Open trial	<p>Outcome measures:</p> <p>Mood symptoms were assessed with the HDRS and YMRS. There were no significant changes on these scales. However levels of pre-treatment symptoms were low.</p> <p>Objective and subjective functioning was rated using the GAF and the Medical Outcomes Survey SF-36. Scores on both measures increased significantly by the end of treatment (14 weeks).</p> <p>In particular the vitality subscale and the role-emotional subscales of the MOS demonstrated significant improvement. There was a trend for improvement on the MOS social functioning subscale.</p>
Scott, J., Garland, A., & Moorhead, S.	2001	A pilot study of cognitive therapy in bipolar disorders	RCT, CBT (n=21) versus 6 months waiting list control followed by CBT. Follow up at 6 and 12 months.	<p>CBT group showed significant reduction in symptomatology and functioning after treatment and follow up.</p> <p>Reduction of depressive symptoms and trend towards fewer manic symptoms.</p> <p>Significantly reduced hospitalisation rates.</p>
Schmitz, J.M., Averill, P., Sayre, S., McCleary, P., Moeller, F.G. & Swann, A.	2002	Cognitive-Behavioural Treatment of Bipolar Disorder and Substance Abuse: A Preliminary Randomized Study	RCT – Medication monitoring (MM) + individual CBT versus MM only	<p>The two groups did not differ in substance use outcomes during treatment, but there was some indication of greater improvement in the MM+CBT group with regard to outcomes related to medication compliance and mood symptoms.</p> <p>Outcome measures – SADS-C, BDI, structured MM interview, urine drug screening, blood sampling.</p> <p>Days reporting depressive and manic symptoms were measured. For depressive symptoms, univariate analyses of variance were significant for treatment condition at 2 weeks. For manic symptoms, significant treatment effects were found at 2, 8, and 12</p>

Lam, D.H., Watkins, E.R., Hayward, P., Bright, J., Wright, K., Kerr, N., Parr-Davis, G. & Sham, P.	2003	A Randomized Controlled Study of Cognitive Therapy for Relapse Prevention for Bipolar Affective Disorder	RCT – Cognitive therapy versus control group (minimal psychiatric care).	<p>weeks.</p> <p>For the MM+CBT group, changes from baseline to end of treatment on measures of mood symptoms revealed improvement on the SADS-C and BDI. These differences were significant for every subscale of the SADS-C except anxiety. The difference was not significant for the BDI.</p> <p>On a treatment satisfaction survey, 75% of MM+CBT patients rated the combination of medication and therapy to be most beneficial compared with each individual component. 75% also perceived the treatment to be most effective for both their bipolar symptoms and substance use problems.</p> <p>Outcome measures – ISS, MRC-SPS, CPI, MCQ, DAS-24, MRS, BDI, BHS.</p> <p>Episodes – The CT group had significantly fewer bipolar episodes, days in a bipolar episode, and number of admissions for this type of episode.</p> <p>Mood questionnaires - During 12 months the CT group showed significantly less mood symptoms on the monthly mood questionnaires. BDI scores of the CT group dropped across time, whereas those of the control group increased with time. No significantly differences between the groups in the mean total BDI, BHS, and ISS activation scores during the whole year, but scores were significantly lower in the CT group at various monthly points. lower on each scale</p> <p>Mood fluctuations – The CT group showed significantly less fluctuation in manic symptoms as measured by the SD of the ISS activation, BDI, and BHS scores.</p> <p>Social functioning, coping, DAS (control) – The CT group showed significantly higher social functioning. The CT group coped significantly better with manic and depressed prodromes at 6 and 12 months and with manic prodromes alone at 12 months. The CT group also scored significantly better on the DAS control subscale at 6 months.</p>
Danielson, C.K., Feeny, N.C.,	2004	Psychosocial Treatment of Bipolar Disorders in	Treatment development and case study	<p>Outcome measures – Inventory of Depressive Symptoms (IDS), YMRS, General Behavior Inventory (GBI).</p>

Findling, R.L. & Youngstrom, E.A.	Adolescents: A Proposed Cognitive-Behavioural Intervention		<p>The patient's symptoms diminished across all measures, with the exception of the YMRS which was zero at both pre- and post-treatment.</p> <p>At 8-week follow-up the patient did not report a return of symptoms (C-BGI scores) but his mother reported a return of some depression and many hypomanic symptoms (P-CBI scores).</p>
Lam, D.H., Hayward, P., Watkins, E.R., Wright, K. & Sham, P.	Relapse Prevention in Patients With Bipolar Disorder: Cognitive Therapy Outcome After 2 Years	2005	<p>The effect of relapse prevention was mainly in the first year – CT had no significantly effect in relapse reduction over the last 18 months.</p> <p>The CT group exhibited significantly better mood ratings, social functioning, coping with prodromes, and dysfunctional goal attainment cognition than the control group.</p> <p>The CT group consistently showed a tendency to perform better than the control group at every time point on all 6 measures (MRS, HRDS, Social Functioning Schedule, Coping with Bipolar Prodromes Schedule – mania and depression, DAS goal attainment).</p> <p>Differences in DAS goal attainment at month 18, social functioning at month 24, coping with mania and depression prodromes at month 24 and mania ratings at month 30 reached statistical significance.</p>
Scott, J., Paykel, E., Morriss, R., Bentall, R., Kinderman, P., Johnson, T., Abbott, R. & Hayhurst, H.	Cognitive-behavioural therapy for severe and recurrent bipolar disorders	2006	<p>Time to recurrence of an episode, based on SCID interview. There were no between group differences in rates of recurrence as a whole, or when depressive and manic recurrences were considered separately. There were no significant differences in duration of each illness episode between treatment groups.</p> <p>Severity of overall symptom levels, based on the Longitudinal Interval Follow-up Evaluation (LIFE). Two LIFE scores were included: mania and depression. There were no significant between-group differences on LIFE scores over 18 months.</p> <p>Post-hoc analysis showed a significant interaction between randomised treatment and number of episodes recorded at baseline assessment. TAU resulted in a slight increase in the proportion who experienced a recurrence (55% - 66%), whereas the CBT group had a steeper increase (41% - 81%). A similar interaction was found with a median split in number of previous episodes – CBT was only of significant benefit to subjects with 12</p>

Ball, J.R., Mitchell, P.B., Corry, J.C., Skillecorn, A., Smith, M. & Malhi, G.S.	2006	A Randomized Controlled Trial of Cognitive Therapy for Bipolar Disorder: Focus on the Long-Term Change	RCT – CT versus TAU. CT modelled to include emotive techniques.	<p>or fewer episodes.</p> <p>Outcome measures included: Relapse rates, dysfunctional attitudes, psychosocial functioning, symptom severity, hopelessness, self-control and medication adherence.</p> <p>Assessments: SCID-IV, DIGS, Structured self-monitoring mood records, serum concentrations, adherence self-report measure, (Social Performance Scale, GAF, Social Adjustment Scale, HAM-D, MADRS, YMRS, BDI, BHS, ISS, ATQ, DAS, SCBS, CGI.</p> <p>At post-treatment, CT patients had experienced significantly less severe depression scores (BDI and Montgomery-Asberg Depression Rating Scale) and less dysfunctional attitudes (DAS). A trend in the same direction was observed for the HAM-D and SPS. The SPS approached significance at 6-month and 9-month follow-up with the CT group experiencing lower levels of disability due to bipolar disorder compared to the TAU group, but this was not maintained or strengthened at 12 months.</p> <p>Both groups improved on a number of measures over the 18-month study period (significant for BDI, BHS, YMRS, ATQ-Positive subscale, DAS, SCBS, SAS).</p> <p>Relapse - no significant differences observed between the groups for number of overall bipolar episodes or singular episode types.</p> <p>After controlling for the presence of major depressive episode at baseline, there was a trend toward a greater time to depressive relapse for the CT group. This trend was not observed for overall bipolar episodes or manic/hypomanic episodes.</p> <p>At 12-month follow-up, the CT group showed a trend toward lower YMRS scores and improved behavioural self-control (SCBS). The Clinical Global Impressions-Improvement scale, comparing the 18 months prior to treatment to the severity of illness status at follow-up, showed a substantial difference between groups in favour of CT. At 12-month follow-up the CT group had significantly greater improvements in the severity of their</p>
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Vasile, D., Vasiliu, O., Ojog, D., Vasile, M., Sarmache, M. & Terpan, M.	2007	Augmenting Antidepressant Psychopharmacological Approach with Cognitive-Behavioural Therapy in Bipolar Depression	Abstract for poster session. Drug treatment only versus drug treatment + CBT (unclear whether patients were randomly allocated into groups, just says they were "distributed in two equally groups").	depressive symptoms and overall bipolar symptoms compared to the TAU group. The difference for mania symptoms approached significance. There were no differences between the groups when improvement in illness severity over the 18-month period of the study was compared to illness severity in the 18-month period preceding the study. No significant differences between the groups on self-reported medication compliance at post-treatment or 12-month follow-up. There was a significant improvement in patients receiving CBT treatment: -12%HAMD, -14%GAF, -16%CGI. YMRS was stable in both groups. The onset of antidepressant action was observed earlier in the CBT group (10.5 days compared to 17.5)
Weiss, R.D., Griffin, M.L., Kolodziej, M.E., Greenfield, S.F., Najavits, L.M., Daley, D.C., Doreau, H.R. & Hennen, J.A.	2007	A Randomized Trial of Integrated Group Therapy versus Group Drug Counselling for Patients with Bipolar Disorder and Substance Dependence	RCT – Integrated group therapy (IGT) versus group drug counselling	Primary outcome measure – number of days of substance use. ASI, timeline follow-back technique, urine toxicology screens. Primary mood outcome – number of weeks ill with a mood episode. LIFE: combination of HAM-D, YMRS, SCID-IV. IGT patients had significantly fewer days of substance use during treatment and follow-up. Groups were similar in the number of weeks ill with bipolar disorder during treatment and follow-up. IGT patients had more depressive and manic symptoms (HAM-D and YMRS) during treatment and follow-up. YMRS scores improved over time during treatment only. HAM-D scores improved during follow-up only. Since number of weeks ill was the same for both groups it is likely that the differences in mood scores represent subsyndromal mood symptoms. It is unclear whether this finding represents true mood differences or an increased level of awareness among IGT patients.
IPT/IPSRT				
Frank, E., Hlastala, S., Ritenour, A., Houck, P., Tu, X.M.,	1997	Inducing Lifestyle Regularity in Recovering Bipolar Disorder Patients: Results from the	RCT – IPSRT versus standard medication clinic treatment (Clinical Status and Symptom	Outcome measures – The Social Rhythm Metric (SRM), the SRM score, HRSD with 8 additional items to assess reverse vegetative symptoms, B-RMS.

Monk, T.H., Mallinger, A.G. & Kupfer, D.J.	Maintenance Therapies in Bipolar Disorder Protocol.	Review Treatment – CSSRT) as adjuncts to standard pharmacotherapy. Subjects are randomly assigned to IPSRT or CSSRT in addition to pharmacotherapy. After at least 12 weeks of treatment and a 4-week period of remission, patients are randomised again into IPSRT or CSSRT groups for 2 years preventative treatment.	Subjects assigned to IPSRT showed significantly greater stability of daily routines with increasing time to treatment. There were no significant differences between the 2 groups in symptomatology.
Frank, E., Swartz, A.H., Mallinger, A.G., Thase, M.E., Waever, E.V. & Kupfer, D.J.	Adjunctive psychotherapy for bipolar disorder: Effects of changing treatment modality.	RCT – IPSRT versus Intensive Clinical Management (ICM). As above study patients were enrolled during an acute episode, randomly assigned to acute phase treatment, then randomly reassigned for preventative treatment.	Outcome measures – Composite score from HRSD with 8 additional items to assess reverse vegetative symptoms and B- RMS; blood levels. Participants remaining in the same treatment for both acute and preventative phases had lower rates of recurrence and levels of symptomatology over the subsequent 1 year period than those reassigned to the alternate modality.
Rucci, P., Frank, E., Kostelnik, B., Fagioli, A., Mallinger, A.G., Swartz, H.A., Thase, M.E., Siegel, L., Wilson, D. & Kupfer, D.J.	Suicide Attempts in Patients with Bipolar I Disorder During Acute and Maintenance Phases of Intensive Treatment with Pharmacotherapy and Adjunctive Psychotherapy	RCT – IPSRT versus ICM (randomised groups for acute and preventative treatment as above).	Outcome measure – suicide attempts, as measured by NIMH-Life- Chart. The group as a whole experienced a threefold reduction in the rate of suicide attempts during the acute treatment phase and a 17.5-fold reduction during maintenance treatment compared with the pre-treatment phase. Both these were significant There were no significant differences in the mode-specific rates.
Frank, E., Kupfer, D.J., Thase, M.E., Mallinger, A.G., Swartz, H.A., Fagioli, A.M., Grochocinski, V., Houck, P., Scott, J., Thompson, W. & Monk, T.	Two-Year Outcomes for Interpersonal and Social Rhythm Therapy in Individuals With Bipolar Disorder	RCT – as above	Main outcome measures: Time to stabilisation in the acute phase Time to recurrence in the maintenance phase assessments used: 17- and 25-item HRSD (8 additional items to assess reverse vegetative symptoms) B-RMS Duke Severity of Illness Checklist Social Rhythm Metric

					<p>Therapy Rating Scale (to evaluate the extent to which sessions focused on intervention-specific themes)</p> <p>No difference between the treatment strategies in time to stabilisation.</p> <p>After controlling for covariates of survival time, IPSRT patients in the acute treatment phase survived longer without a new affective episode, irrespective of maintenance treatment.</p> <p>IPSRT patients had higher regularity of social rhythms at the end of acute treatment.</p>
Swartz, H.A., Pilkonis, P.A., Frank, E., Proietti, J.M. & Scott, J.	2005	Acute Treatment Outcomes in Patients with Bipolar I Disorder and Co-Morbid Borderline Personality Disorder Receiving Medication and Psychotherapy			

IV A cognitive interpersonal approach to recovery and relapse prevention

4.1 CBT for Bipolar Disorders

Overall a cognitive behavioural approach to the treatment of bipolar disorder is aimed at enhancing non-pharmacological coping skills, to enhance elements of self-efficacy and responsibility in the treatment of the condition, to support individuals in recognising and managing psychosocial stressors and the impact of past episodes, to introduce specific strategies to deal with cognitive and behavioural difficulties, and to modify underlying schemata and core assumptions.

CBT for bipolar disorder relies on the basic characteristics of a CBT model, in that the cognitive behavioural model is most effective when the individuals are full collaborative partners in the treatment process. The therapist educates the individual about the diathesis-stress model of bipolar disorder, socialises the individual into the cognitive model of mood changes, and appraises them of the rationale for particular interventions. An assessment of the individual's core beliefs and underlying schemata are essential in the case formulation of individual vulnerabilities that form an integral part of the treatment plan.

The CBT treatment of bipolar disorder is naturally phase specific. The specific focus of the intervention will vary depending on the individual formulate on of treatment goals

and the phase of the disorder in which the patient presents. For example if a patient presents in an acute phase of a bipolar episode the cognitive behavioural strategies will be aimed at crisis intervention, the treatment of acute symptoms, an assessment of risk and factors that are maintaining the episode, and the establishment of a good therapeutic alliance. If a patient presents in the recovery phase following a recent episode or in a phase of stabilisation between episodes, the CBT treatment would intend to be insight oriented, to explore the meaning and context of symptoms, interpersonal functioning, preventative cognitive strategies, and self-management skills, to reduce the impact psychosocial of the disorder and to build resilience regarding ongoing stressors.

The following section is going to outline the four main components of cognitive behavioural psychotherapy for bipolar disorder: Psycho-education, early warning signs and coping with prodromal symptoms, cognitive behavioural strategies for dealing with manic, hypomanic or depressive symptoms, and finally the targeting of associated difficulties in psychosocial functioning, especially interpersonal difficulties.

It seems within recent developments for complex disorder groups, e.g. personality disorders, psychosis and recurrent mood disorders, that there is an emphasis on the development of an individual case formulation for the adaptation of a cognitive behavioural intervention to any specific disorder group. This should be developed in collaboration with the patient and it should be based on a developmental and cognitive model of the specific phenomenology of the bipolar disorder. The cognitive formulation is the starting point for the therapeutic intervention and can be used as an alternative explanation of the patient's difficulties and will help to engage the patient into a cognitive way of understanding and working with the presenting problems.

4.1.1 Self monitoring and prodromal changes

Dealing with manic and hypomanic phases has been described as being the biggest clinical challenge in the treatment of bipolar individuals. Most individuals suffering from bipolar disorder would describe a manic phase as being inescapable, once their mood starts to rise the initial positive reinforcement of experiencing new sources of energy and creativity develops, especially when this happens after long periods of depressed mood it easily develops into a self-reinforcing pattern that seems impossible to stop.

The psycho-educational component of the cognitive behavioural intervention is an important starting point in this stage of problematic mood changes. The individual's awareness of possible consequences and manic episodes developing in a way that they require increased external control and medications seems crucial in preventing the negative impact of full blown manic episodes. Past episodes provide the best source for information

The early warning signs paradigm, originally developed for relapse prevention in early onset psychotic disorders, especially schizophrenia, has been adapted for the use with people suffering from bipolar disorders (Lam & Wong, 1997). Patients learn to identify prodromal and early symptoms of relapse and develop a range of behavioural techniques to improve their coping skills in order to counteract early symptoms effectively and to avoid their development into a full blown episode.

In most cases the change in mood, cognition and behaviour is a gradual process. This allows time for the clinician and the individual to utilise psychological interventions whilst he or she is still responsive to cognitive and behavioural techniques. Teaching patients to

recognise early symptoms of psychotic relapse and seek early treatment is associated with important clinical improvements (Perry, et al., 1999). Recent advances in the identification and formulation of individualised early warnings signs (Lam & Wong, 1997) and the prodromal 'relapse signature' (Smith & Tarrier, 1992) allows clinicians to reformulate the process of cycling into mania as an interaction of the individual's life situation, cognitive processing and their general level of coping skills. We can help patients to develop an individualised profile of prodromal changes and to be sensitised to significant mood changes early enough to curtail vicious cycles. This therapeutic step is influenced by the idiosyncratic beliefs that each patient associates with changes in their mood and that might compromise their coping abilities in the face of prodromal changes. For example the patient who believes that his or her manic episodes are follow a predetermined course no matter what he or she does, might well be less cautious and responsible in the face of early hypomanic mood changes and therefore exacerbate the development of manic symptoms. These maladaptive beliefs underlying the individual's coping strategies and reactions are crucial especially in the prevention of manic episodes. In utilising cognitive therapy strategies such as cognitive reframing and guided discovery, patients can learn to view new behaviours as an active process in which they execute a choice and that despite the undeniable attraction of hypomanic impulses some degree of control could be established.

One of the difficulties described by many patients is that of developing a hyper-vigilance regarding minor changes in mood and their misinterpretation as onset of a manic episode rather than an accurate reflection of ordinary happiness, which can lead to inappropriate safety behaviours and avoidance. Within a cognitive behavioural framework this can be avoided by teaching the patient to monitor their mood on an ongoing basis using individualised mood monitoring tools that allow the patient to look out for several

specific prodromal signs in connection with actual environmental stressors and events, in order to avoid the generalisation of mood changes. Further to employ coping strategies in response to prodromal changes that are appropriate to the mood changes observed. These coping strategies include activity schedules, the observation of sleep and dietary routines, the practice of relaxation exercises and graded task assignments, time delay rules and problem solving techniques in the face of impulsive decision making, and stimulus control techniques, such as the regulation of alcohol and caffeine consumption, and the reduction of risk seeking behaviours and stimulating activities. Jones argues in a review discussing the benefits of cognitive behavioural interventions for individuals suffering bipolar disorder that the indicated mechanisms of change over and above the known benefits of cognitive therapy indicate behavioural techniques such as extended activity scheduling and stabilisation of daily routines and sleep cycles, that predominantly influence circadian rhythm (Jones, 2001).

Disruption and irregularity in circadian rhythms, social events and activities have been found to significantly impact on mood and can trigger affective episodes in people suffering from bipolar disorders. In support of this effect the regulation of social interactions and balanced sleep wake cycles have been found to be effective in preventing relapse and subsyndromal mood swings in bipolar disorders. Bipolar patients are highly sensitive to disruptions in their biological rhythms (Malkoff-Schwartz, et al., 1998). The regularity of daily routines and activities, as well as the regularity of sleep-wake cycles has been identified as a major protective factor (Frank, et al., 1999). The psychological factors that influence individuals' ability to maintain stability, such as advance planning, attention to detail, and self-restraint, are the very difficulties that are associated with bipolarity. The therapist must therefore be very cautious in introducing these ideas that might easily be perceived as being overly controlling and meet significant resistance from

the patient. One way to evaluate whether positive mood changes are indicative of a hypomanic or manic episode is to engage in calming activities and 'time out' as a way of self-assessment as to whether it is possible for the patient to remain still and to concentrate for significant periods of time.

The most effective intervention towards the successful coping with prodromal symptoms and counteracting mood changes is to re-evaluate the experience of past episodes and their consequences and to engage in a cost benefit analysis of letting things take their natural course or to engage in constructive self-monitoring and self-regulating strategies. A useful therapeutic step within that is the acknowledgement of the difficulties to resist especially hypomanic mood changes and the initial gratification that goes along with it. In this we need to bear in mind that both appraisals of current symptoms as well as the memory of past episodes is influenced by mood congruent biases. It is therefore valuable to use life charting techniques and diary keeping to encourage patients to process recent changes in the context of past experience and in interaction with other life changes (Basco & Rush, 1996).

4.1.2 Cognitive strategies

The cognitive therapy techniques used for bipolar disorders include strategies aimed at the processing of symptoms and cognitive distortions relating to hypomanic and manic episodes. Further it aims to address beliefs and attributional biases linked to the psychological effects of long term impairment through chronic mood related difficulties and/ or residual symptoms.

Most patients suffering from bipolar disorders describe mood related difficulties and their social and interpersonal consequences as dating back to early adolescence. The longstanding nature of many of the associated difficulties and variation in intensity and severity over time makes it difficult for many patients to identify areas of normal functioning or the clear demarcations of the 'healthy self'. Some schema work can therefore prove to be extremely useful in re-examining the value and evidence for old belief systems and the generation of new sets of beliefs adaptive to the current actuality.

Cognitive therapy follows a constructionist view of reality as being created by the individual's idiosyncratic pre-conceptions, perceptions and memories. Cognitive therapy strategies, in the face of significant emotional difficulties, take into account the systematic distortions and maladaptation that can significantly influence the individual patients' world view. This approach aims at the correction or re-evaluation of these systematic mood congruent biases by re-examination of actual experiences and current interpersonal interactions, including the therapeutic relationship (e.g. Newman, et al., 2002). In the presence of signs of mania and hypomania cognitive therapists would aim at helping the patient to reality test and re-examine their extremely positive world view and self perception taking into consideration their current interactions and environmental stressors. Similar micro techniques and strategies come into play for example in the observance of daily thought records. Systematic thinking errors driven by hyperpositive automatic thought patterns and beliefs not unlike the ones observed in depression but with the opposite valence, such as overgeneralisation, mind-reading, and personalisation. In the re-evaluation of these thought patterns it is important for the therapist to support patients in the process of rationalising by emphasising the maladaptive nature of such styles and consideration of likely consequences of hyperpositive thinking.

Especially for manic or hypomanic patients, these attempts might be perceived as extremely counterintuitive and controlling in the light of their self-perception of enjoying life and their new found energy. It is therefore important for cognitive therapists working with bipolar patients to aim at preserving their sense of autonomy, self-efficacy and control over their own lives. Techniques that support the self efficacy and the re-evaluation of maladaptive beliefs include behavioural experiments, the feedback of close others and anticipatory problem-solving.

Patients can be encouraged to test out their assumptions by creating real-life experiments. In hypomanic patients this technique could lead to some reckless behaviour when hyperpositive thoughts are put to the test. In hypomania, therefore, behavioural experiments can be constructed to test out the assumed consequences of not following impulses acting with caution and time-delays. To make constructive use of their social support system bipolar patients often have to meet previous agreements with significant others regarding their intervention and advice, as hypomanic individuals often do not appreciate the influence of others.

One of the main features of manic or hypomanic phases is excessive risk taking. This is accompanied by a set of cognitive biases that leads many bipolar patients to underestimate the potential harm or overestimate the potential benefits of their behaviours (Leahy, 1999). Newman and colleagues (2002) introduce a version of the cost benefit sheets often employed in CBT problem solving techniques to get bipolar patients to balance risk and benefit of actions prospectively; the 'productive potential versus destructive risk rating technique'. In this technique patients use a two column table balancing the 'productive potential' and the 'destructive potential' with the support of the

therapist, which should allow individuals in a hypomanic or manic phase to consider the potentially negative consequences of their actions for others.

Related to these techniques which attempt to help bipolar patients to re-evaluate their hyperpositive thoughts are the following CBT applications to moderate their impulsivity. One example for this is the 'time delay' rule, encompassing contracted agreements to delay the execution of 'spontaneous' ideas, that might include adventurous activities or large purchases. The CBT technique of scheduling daily activities is commonly used to help depressed patients to master day to day activities and to reactivate the enjoyment of favourite past-times; for bipolar patients this technique can be employed to slow down the vicious cycle of mania driven by excessive activities, poor decision making and more poorly deliberated and ineffective activities. Anticipatory problem-solving regarding early warning signs of imminent mood swings and in relation to life stressors that might exacerbate symptoms (Johnson & Miller, 1997) appear to be crucial in these two areas where the coping abilities of bipolar patients can be particularly challenged. Therapeutically the process of anticipatory problem-solving includes the retrospective evaluation of past crises, to identify potential problem areas in major life domains, and using problem solving techniques to deal with these problem areas and obstacles in advance. Another technique to moderate hypomanic and manic mood is stimulus control. This includes the ability to moderate drug and alcohol use, not to engage in extreme sports and other risk taking and 'exciting' activities. Medium to long term choices in this connection include the regulation of working patterns that do not include extreme hours and frequent disruptions of sleep cycles. These strategies, especially when viewed medium to long-term might seem very challenging to individuals who are prone to act impulsively and like to engage in activities without much prior consideration

and planning. To avoid conflict with the high autonomy of bipolar patients the therapist needs to aim to take as collaborative a position as possible.

Many bipolar patients argue that in particular their high moods, euphoria and heightened irritability are autonomous from their volition. Therapeutically it can be extremely challenging to moderate these mood states and to increase the patient's willingness to participate in interventions that are incongruent with their current mood. Cognitive behavioural techniques that can be applied in that context are relaxation and breathing exercises, cognitive strategies to compare the lasting effects of pleasant affective states versus their intensity, and the appraisal of positive beliefs that are linked to the high feelings themselves.

Individuals with bipolar disorder experience frequent and prolonged periods of depression which over time fosters feelings of hopelessness strongly associated with suicidal thinking and suicide. This is seen as being directly related to the problems created by frequent mood swings and associated behaviours. Bipolar patients frequently have to reassemble their lives after episodes of manic acting out and depressive withdrawal, they find it difficult to trust their euthymic mood and not to worry about the impending relapse. The diagnosis itself, its cyclical episodes and their treatments are further associated with stigma and shame, which makes it harder for individuals to utilise and maintain their social support network and prolongs their depressogenic beliefs and hence their vulnerability for relapse (Lundin, 1998). In sum bipolar disorder contains painful and unstable affect, extremes of cognitions and behaviours, interpersonal deficits, and a lasting sense of Sisyphus' despairing exhaustion. As a result the lifetime suicide rates have been found to be between 15 and 25% (Goodwin & Jamison, 1990; Simpson &

Jamison, 1999). An assessment of risk therefore needs to be an ongoing feature in the treatment of individuals suffering from bipolar disorder.

The cognitive behavioural treatment of depression is discussed in detail elsewhere, conceptually it can be applied well to the depressed mood states within bipolar disorder. Here I would only like to point to a few specific aspects that might be more specifically relevant to individuals suffering from bipolar disorder.

Many people suffering from bipolar disorders report a long history of several significant illness episodes, the traumatic impact of multiple hospital admissions and partially successful treatment regimes involving several different psychotropic medications. Individuals in this disorder group often suffer from significant residual symptoms and have experienced short periods of remission followed by frequent relapses. This poses a particular challenge to the clinician; the patient and their significant others might express increased hopelessness regarding remission and scepticism regarding the model offered by the clinician. Key characteristics of chronic or partially remitted disorders, such as suicidal ideation, hopelessness, low self-esteem and self-efficacy, avoidant coping strategies, and poor problem solving are amenable to change utilising cognitive behavioural strategies.

In a high risk population such as patients with bipolar disorder it is advisable to negotiate an anti-suicide agreement, and although such contracts do not prevent suicides they highlight and validate the importance of a safe environment for patients and therapists alike (Stanford, et al., 1994; Kleepsies & Dettmer, 2000). In the face of intense suicidal ideation the therapist aims to reveal the beliefs underlying suicidal thoughts and to engage the patient in the exploration of alternative and life-affirming beliefs. These interventions

include the open investigation of the pros and cons of suicide, the gentle challenging of assumptions behind suicidal thoughts (e.g. suicide as solution to all problems), and consideration of the social context and the consequences of such thoughts and actions. As utilised in the cognitive behavioural treatment of unipolar depression the increase of mastery and pleasure in productive and enjoyable activities can instil hope and encourage self efficacy. Cognitive factors associated with increased risk of suicidality are 'cognitive rigidity', perfectionism, and poor autobiographical recall (Ellis & Ratcliff, 1986; Evans, et al., 1992; Blatt, 1995; Scott, et al., 2000). Cognitive rigidity refers to depressogenic all or nothing thinking and has a strong link with hopelessness and despair associated with suicidality. This particular thinking style is therefore at the core for cognitive interventions. Likewise, perfectionism describes a set of beliefs that makes individuals vulnerable to depression and hopelessness, and it compromises constructive problem-solving. Zuroff and colleagues (2000) suggest that perfectionist beliefs are related to self-criticism, perceived stress, increased interpersonal problems, and they can further impede the therapeutic alliance. Poor autobiographical recall has been linked to problem-solving deficits in unipolar and bipolar depressed individuals (Evans, et al., 1992; Scott, et al., 2000), it compromised the individuals' ability to learn from past experience and it can thus chronify old dysfunctional beliefs.

Central to the effective treatment of chronic or acute depressive difficulties in bipolar patients is the optimal utilisation of their social support network. The consequences and interactional styles of both manic and depressed episodes can easily compromise the individuals' relationships. A careful assessment of the individuals' social network and the relationships that survived following many mania-induced conflicts and depression-induced estrangements will provide a fruitful starting point for the rebuilding of a stable and supportive social environment. Detailed analysis of specific interactions or situations

as well as role-playing and other social skills training techniques might provide crucial assets in the cognitive behavioural intervention.

4.1.3 Interpersonal functioning

The third phase of the treatment is targeted towards the interpersonal difficulties that precipitate or resulted from the disorder. This is where cognitive strategies address core beliefs and schemata. The goals for this phase of the treatment include the experience of increased self-efficacy and the rebuilding of a more solid and autonomous sense of self. This takes account of the impact of the illness which often occurs in a developmentally critical time when self-esteem and identity are formed. It further appears that the impact of mania and depression at an early age are significant as they dramatically affect important developmental milestones such as educational achievements, early work experience and important interpersonal relationships. Essential cognitive structures such as dysfunctional core beliefs will likely become self-perpetuating. Examples of these beliefs include a distorted sense of autonomy, or personal capability, vulnerability to harm or illness, and a sense of defectiveness and unlovability. The recognition of maladaptive core beliefs that may have been established by the early onset of the disorder or traumatic events are important to address as it will help those individuals to understand and cope with the specific psychosocial impairments experienced later in the life course.

These interpersonal vulnerabilities and risk factors can play a major part in the recovery and prevention of relapse of the individual. Therapeutically some of this process will consist of the facilitation of successful transitions following major episodes, significant psychosocial changes and the adjustment to necessary life-style changes. Similar to the above mentioned model of the importance of corrective experiences and behaviour

change in individuals with bipolar disorder, these changes in the cognitive emotional schemata of the bipolar patient are achieved through consistent behavioural adaptations to the vulnerabilities intrinsic to the disorder. In their reformulation of the interpersonal psychotherapy framework (IPT) for bipolar disorder (IP/SRT) Frank and colleagues (1997) combine the key interpersonal difficulties associated with bipolar disorder with an introduction of the strict monitoring of social routines and circadian rhythms. By addressing interpersonal problems and the regularity of daily routines, this method addresses both concurrent symptoms and the impact of interpersonally based stressors on the patient's life, and increases his or her resilience to potential vulnerabilities.

The application of these techniques within an integrated psychological therapy framework allows the patient to develop an understanding of how adverse interpersonal experiences create maladaptive schemata about the self, dysfunctional attachment beliefs and impair the acquisition of effective interpersonal problem solving strategies. The individual also gains insight how these might alter the threshold of stress needed to trigger a depressive or manic reaction, and how the generation of these events might be maintained by dysfunctional ways of solving emerging interpersonal difficulties and from conflicts arising from maladaptive expectations about others (Lovejoy & Steuerwald, 1997). The direct therapeutic targeting of these interpersonal vulnerabilities can lead to schema change and the development of stable supportive interactions in the presence of negative life events that aid the prevention of relapse.

4.2 Psychological factors in vulnerability to relapse

4.2.1 Affect, meaning and relapse

A key aspect of relapse is the experience of high levels of emotional distress and affective dysregulation in the period before, during and following an acute episode. This has long been recognised by researchers and clinicians alike. For example, Docherty and colleagues (1978) proposed that prior to the development of a full blown relapse there were identifiable and sequential phases, which they saw as an unfolding of a series of psychological states. These phases were conceptualised as being characterised by feelings of overextension, restricted consciousness, behavioural and affective disinhibition, psychotic disorganisation and resolution. During the first phase of overextension, the person experiences a sense of being overwhelmed by stressful demands or internal / external conflicts and is accompanied by feelings of fear, threat, anxiety and nervousness. This phase is followed by the appearance of a variety of intrusive mental phenomena, which limit the person's ability to concentrate and think. The person experiences feelings of helplessness, hopelessness, dissatisfaction, and loneliness. During the disinhibition phase, the capacity of the individual to modulate or regulate their internal impulses becomes impaired. The signs and symptoms of this phase are rage, panic and hypomania. This precedes increasing perceptual and cognitive disorganisation, loss of self-identity and fragmentation of control during the active phase of psychosis. Docherty and colleagues' formulation emphasises a sequential view of the nature of relapse, where relapse is characterised by the progression of increasing non-psychotic symptoms, through increased emotional distress, affective dysregulation, psychological fragmentation, feelings of loss of control, culminating in the evolution of psychosis. The importance of the role of affect in psychotic relapse has been consistently demonstrated in a number of

retrospective and prospective studies examining the prediction of relapse itself. Retrospective studies of individuals and their families (Herz & Melville, 1980; McCandless-Glimcher et al., 1986; Birchwood et al., 1989) show that the most commonly reported early signs of relapse are fearfulness, anxiety, poor sleep, irritability, tension, depression, and social withdrawal.

The consistency with which early signs of relapse have been reported has led to the development of prospective investigations of early signs. In essence, these studies have sought to identify the sensitivity and specificity of these early signs as an indicator of emerging relapse. Clearly if these early signs are sensitive and specific to relapse, the monitoring of such signs, and associated emotional distress, would help facilitate earlier interventions - potentially leading to the prevention and/ or amelioration of relapse. In the investigation of the predictive power of early signs monitoring, *sensitivity* refers to the ability of the monitoring system to correctly identify a forthcoming relapse. It is essentially the proportion of individuals who experience early signs prior to a relapse. *Specificity* refers to the power of these early signs to correctly identify those individuals or times when a relapse will not occur.

Subotnik and Neuchterlein (1988) reported a prospective study of early signs in relation to relapse amongst 50 individuals. Participants were monitored fortnightly and relapse was defined by a rating of severe or extremely severe on the Brief Psychiatric Rating Scale (BPRS) Unusual Thought Content, Conceptual Disorganization, and/ or Hallucinations items. Greater suspiciousness and thought disturbance symptoms correctly identified 10 out of the 17 relapses. This gave a sensitivity to relapse of 59%. Birchwood and colleagues (1989) recruited 17 individuals participants who were monitored fortnightly using the self rated or observer rated Early Signs Scale over a nine-month

period. Relapse was defined as any hospital admission or a clinician's judgment of imminent relapse or probable admission. Eighty two percent of those who experienced a relapse had an increase in early signs prior to relapse. Sixty two per cent of those who did show an increase in early signs went on to have a relapse, meaning that 38% had an increase in early signs but did not go on to relapse. Tarrier et al., (1991) monitored 56 participants on a monthly basis. Relapse was defined as a reappearance of positive psychotic symptoms or the worsening of persistent or residual positive symptoms, which lasted for at least one-week. Depressed mood alone was associated with a sensitivity of 50% and specificity value of 81%. When depression was combined with hallucinations, the sensitivity value increased to 62.5% and the specificity value was 87.5%. Malla and Norman (1994) monitored 55 participants on a monthly basis over a period of at least 12 months (range: 12-29 months). In this study many increases in psychotic experiences were not preceded by increases in emotional distress, unless accompanied by increases in psychotic symptoms. Jorgensen (1998) monitored 60 individuals, 30 of whom had residual positive psychotic symptoms ("symptomatic"), and 30 who were fully remitted ("asymptomatic"). Participants were interviewed every fortnight over six months or to relapse. In total, 45% participants relapsed, 27% of whom were readmitted to hospital. For symptomatic participants sensitivity of early signs to relapse was 88% and specificity 64%, and for asymptomatic participants the sensitivity value was 73% and the specificity value was 89%. Across the eight studies reporting sensitivity and specificity for early signs to relapse, the findings on sensitivity values range from 8 to 88%, and for specificity values from 64 to 93%. Strict comparison across these studies is problematic given the nature of differences in methodology and design. However a number of conclusions are possible on the basis of these data.

Whilst it is not easy to group together studies examining observer and studies examining self rated early signs due to important methodological differences, it is noteworthy that the median sensitivity of observer rated early signs (Subotnik & Neuchterlein, 1988; Tarrier et al., 1991; Gaebel et al., 1993; Marder et al., 1994) was 37%, whilst for those studies incorporating self rated early signs monitoring (Birchwood et al., 1989; Malla & Norman, 1994; Jorgensen, 1998; Hirsch & Jolley, 1989) the median sensitivity results were 68%. As the reader will recall, sensitivity refers to the ability of early signs to correctly identify a forthcoming relapse. Therefore, in this case self rated early signs seem much more powerful in predicting relapse, suggesting that individuals' unique knowledge of their own experiences gives them a better ability to predict relapse than the health professionals that provide support and treatment for them. This also means that it is likely that individuals are detecting their own idiosyncratic signs of relapse at an early stage. Depending on their experiences of previous episodes relapse / psychosis this is likely to generate a high degree of emotional distress.

It is also very apparent that when studies include positive psychotic experiences or incipient psychosis in their definitions of early signs (Subotnik & Nuechterlein, 1988; Birchwood et al., 1989; Tarrier et al., 1991; Jorgensen, 1998), this increases the sensitivity of early signs detection to relapse. The inclusion of low level positive psychotic symptoms, such as ideas of reference or thought control, suggests that the development of emotional distress signals the person's emotional reaction to the reemergence of psychotic experiences. The consistency of the findings on specificity of early signs to relapse, which is reported across these studies (64 to 93%), is supportive of this proposal. That is, when there is a relapse there is almost always an increase in emotional distress before hand.

It is likely that individuals may well be responding to quite subtle changes in their cognition, perception, and attention that are psychologically significant or reminiscent of psychosis. Early studies (e.g. McGhie & Chapman, 1961; Chapman & McGhie, 1963; Freedman & Chapman, 1973; Docherty et al., 1978; Henricks et al., 1985) found in clinical interviews that idiosyncratic changes in the perception of cognition, emotion and interpersonal experience appeared to be associated with psychosis, and that these experiences are different to those whose psychosis has remitted or those suffering from depression (Cutting, 1985). Chapman and McGhie (1963) suggested that individuals with psychosis become aware of unusual experiences, and that their reactions to these experiences may play an important role in the development and maintenance of psychosis. They recommended that a psychotherapeutic understanding of the individual's perceptual and experiential difficulties would aid improved communication. In addition, they suggested that psychotherapy should aim to (a) discover individuals' subjective experiences and cognitive difficulties, and (b) reduce unhelpful or ineffective reactions to these experiences. Bowers (1968) argued that self-experienced changes in perception and awareness were critical to the transformation of normal experience into psychosis. In an experiential account drawn from interviews with fifteen people with psychosis, Bowers described changes in heightened awareness of internal and external stimuli. Associated with these perceptual changes he described individuals as having an increasing sense of urgency, reduced need for sleep, exaggerated affect, and a heightened sense of self. Alongside this heightened experience, internal and external events and stimuli normally outside awareness became meaningful and self-relevant. Individuals described becoming engaged, fascinated, perplexed or indeed scared by their own experience.

4.2.2 A cognitive behavioural model of early signs and relapse

There have been a number of psychological conceptualisations of relapse (Thurm & Haefner, 1987; Birchwood, 1995; Gumley et al., 1999). All of these models have emphasised how individuals interpret subtle signs (e.g. cognitive perceptual changes) and / or symptoms (e.g. interpersonal sensitivity) as evidence of a forthcoming relapse of their psychosis. In this context individuals' interpretations of their experiences will be informed by their specific autobiographical memories (of psychosis). For some individuals who do not accept the construct of psychosis or illness, these signs may signal elevated interpersonal danger (e.g. "if my doctor sees that I'm suspicious he'll put me in hospital again"). These memories and appraisals drive the development of heightened emotional distress and trigger affective dysregulation. Coping strategies adopted by individuals may enable them to reduce their levels of emotional distress or support affective stabilisation. For example, being able to talk with a trusted friend or family member, being able to self-soothe, having a kindly, accepting and compassionate attitude to oneself, being able to decatastrophise relapse, or being able to access appropriate support and assistance available may all positively impact on coping. Three studies (Brier & Strauss, 1983; McCandless-Glimcher et al., 1986; Hultman et al., 1997) show that patients monitor and regulate their symptoms in order to prevent relapse. Amongst individuals with Bipolar Disorder, Lam et al., (1997, 2001) reported on the use of spontaneous cognitive and behavioural coping strategies during prodromal stages, and also the impact that these had on functioning. They reported that the use of behavioural coping strategies had an effect on reducing the likelihood of manic relapse. On the other hand, having few interpersonal resources, living in a highly stressful environment or being socially isolated may well limit the availability and flexibility of coping strategies or the opportunities for help seeking.

The use of coping strategies such as substance use or medication discontinuation (to reduce side effects) may provide short term relief but enhance relapse risk in the medium and long term. Social avoidance and withdrawal may enhance interpersonal sensitivity, rumination and emotional distress leading to feelings of helplessness, hopelessness and suicidal thinking. Hultman et al., (1997) found that individuals with a withdrawal-orientated coping style were more likely to relapse than individuals who had a socially orientated coping style. In addition, problematic thought control strategies or avoidance strategies may prevent disconfirmation of excessively negative beliefs about relapse thus maintaining (a) an elevated sense of threat of relapse and (b) increasing the likelihood of greater relapse acceleration at the appearance of early signs.

Safety behaviours are a kind of coping strategy specifically targeted at attempting to avoid a feared outcome. Not only do these behaviours attempt to avert a feared outcome, they also prevent the individual from disconfirming unhelpful beliefs, and thus play a role in the maintenance of anxiety and psychological distress.

Prospective studies of the course and outcome following a first episode of psychosis have consistently shown that poorer outcome, as characterised by treatment resistant symptoms or relapsing course of psychosis, is predicted by factors such as longer duration of untreated psychosis (DUP; Drake et al., 2000; Weirisma et al., 1998), poor pre-morbid social adjustment (e.g. Rabiner et al., 1986), early adolescent isolation (e.g. Robinson et al., 1999), and adolescent social anxiety (Jones et al., 1994). Of course we need to remain mindful of the catch that developmental reasoning can pose for the transition of these findings into clinical work; *“so long as we trace the development from its final outcome backwards, the chain of events appears continuous, and we feel we have gained an insight which is completely satisfactory or even exhaustive. But if we proceed in the reverse*

way, if we start from the premises inferred from the analysis and try to follow these up to the final results, then we no longer get the impression of an inevitable sequence of events which could not have otherwise been determined” Freud (1920, p. 208).

Drake and his colleagues (2000) found that after controlling for DUP, better levels of social integration predicted greater improvement in overall symptoms including psychotic experiences following a first or second episode of psychosis. These data suggest that early social/ developmental factors and adolescent or adult interpersonal functioning are powerful potentiators of outcome in terms of recovery and relapse following the onset of psychosis. In particular, it appears that the quality of adolescent peer attachments and utilisation of social supports strongly predict longer term outcomes and adaptation to psychosis. There is also extensive evidence from studies of schizophrenia that the interpersonal atmosphere is a key factor in relapse (Brown & Rutter, 1966, Leff & Vaughan, 1985). This demonstrates the continuing significance of interpersonal factors in the determination of course of psychosis. Furthermore, after a first episode of psychosis the recurrence of psychotic experiences is often a highly significant, distressing and critical life event for individuals with psychosis (Birchwood et al., 2000) leading to problematic emotional adjustment and suicidal thinking. A number of studies have now shown that the way in which the early episodes of acute psychosis are experienced and processed by the person can have a significant influence over recovery and relapse. How the experience of psychosis is appraised has been linked with a variety of important emotional responses in individuals, families, carers and loved ones. Often these responses do not just relate to a person's current experience of a psychotic episode but these responses are essentially reflections of particular early experiences, general developmental factors and their social context.

In terms of the interpersonal context of relapse and recurrence of episodes Expressed Emotion is a key concept to conceptualise the impact of close social environments on the adjustment and recovery of the individual. The EE literature has been reviewed in detail in Chapter 2. Beyond a behavioural and interactional model of EE, Barrowclough and colleagues (1994) investigated the role of relatives' attributions for illness as a predictor of relapse. Barrowclough and her colleagues proposed that critical and in particular hostile relatives would make attributions, which were more internal, personal and controllable to the individual, compared to relatives who display marked EOI and low EE patterns. They proposed that these latter relatives would make more external, universal, and uncontrollable attributions to the individual. Relatives with high levels of EE had a higher rate of making causal attributions than did relatives with low EE. Compared to high EOI relatives, relatives high on criticism and hostility made more internal, personal and controllable attributions to the individual. In addition, these relatives invoked causal attributions attributing responsibility for outcome to the individual. Relatives high on EOI, made more attributions of causality to external and uncontrollable causes, and indeed made most attributions to illness. Attributions of controllability and internality (e.g. it's your fault) were significantly related to relapse, even after controlling for EE status and intervention.

These studies demonstrate the importance of the role of attributions in the development of expressed emotion, the prediction of relapse and the development of distress amongst relatives. Indeed this is consistent with the proposals of other investigators including Weiner (1985) who proposed that causal beliefs held about other people's problems would be instrumental in the development of distressing emotions. Barrowclough and colleagues (2003) explored the relationship between interpersonal environment, self-evaluation and positive and negative symptoms. Whilst it is well established that living in

an emotional climate characterised by critical, hostile or emotionally over-involved attitudes and attributions is linked to increased risk of future relapse (Barrowclough & Hooley, 2003), the mechanism by which this might occur is as yet unclear, although increased physiological arousal has been proposed as one pathway (Tarrier & Turpin, 1992). Using an interview methodology with a group of 59 participants, Barrowclough and colleagues found that, consistent with previous research, family attitudes were associated with greater positive symptoms. In addition, participants' negative evaluations of self (NES) were associated with more positive symptoms. The relationship between family attitudes and positive symptoms disappeared when NES was included in the statistical model, and NES remained strongly associated with positive symptoms. Therefore, in this sample the impact of criticism on participants' positive symptoms appeared to be mediated by participants' own negative self-evaluation.

Surprisingly, there has been little investigation into the separate developmental pathways of criticism and emotional over-involvement for the purposes of early intervention and support. In a study by Scazufca and Kuipers (1996) changes in expressed emotion were linked to changes in the subjective burden and perception of a client's functioning. This supports the notion that expressed emotion is not a trait characteristic but varies according to patient-relative interactions at a particular point in time (Birchwood & Cochrane, 1990). Existing family intervention methods designed for use with families experiencing the effects of a longer-term psychosis in a relative appear to be less useful in first episode samples. One study of behavioural family therapy in first episode psychosis found no impact on relapse and that for some families it actually increased distress (Linszen & Dingemans, 1996).

Emotional over-involvement is seen more often in parents and has been linked to the individual's poor pre-morbid functioning and burden (Miklowitz et al., 1983). Miklowitz and colleagues (1983) found that over involved family members tended to have relatives with poor premorbid histories and many residual symptoms in comparison to both critical family members and low expressed emotion family members. Cook, Strachan, Goldstein & Miklowitz (1989) found that adolescents interacted with high expressed emotion mothers more problematically than adolescents with low expressed emotion mothers. High expressed emotion mothers then tended to reciprocate negative affect of their adolescents interactional style. In this sense we can understand the evolution of expressed emotional as a reciprocal process in the context of adolescent development. Dozier and colleagues (1992) have suggested that the attachment strategies of individuals themselves may be important in eliciting characteristic expressed emotion responses from their family members. They investigated attachment organisation and expressed emotion of family members amongst 40 participants with schizophrenia ($n = 21$) and affective disorders, mainly bipolar disorder ($n = 19$). More extreme use of avoidant or pre-occupied attachment strategies was associated with higher levels of emotional overinvolvement of family members. It may be that greater reporting of distress and neediness which is characteristic of a pre-occupied attachment style may elicit more caring behaviours by family members. Avoidant strategies tended to be associated with lower symptom reporting and self reported distress. Dozier and colleagues noted that this strategy may be unconvincing to family members, and therefore families sense the need to provide care. Emotionally over involved strategies may then perpetuate problematic attachment strategies either by reinforcing feelings of neediness and vulnerability in preoccupied persons or driving greater withdrawal and avoidance in individuals who use avoidant attachment strategies. These findings highlight the importance of working with individuals' interpersonal and attachment strategies in the

context of living in a high expressed emotion environment. This may be particularly useful in scenarios where patients and / or relatives decline to participate in family based interventions.

4.2.3 Trauma

Morrison and colleagues (2003) have argued that rates of childhood trauma are elevated amongst individuals with psychosis. This is based on several lines of evidence. First, studies have demonstrated that there is a high rate of trauma in the lifetimes of those individuals who have established psychosis. For example in a casenote review of 200 individuals, Read and colleagues (2003) found that those who had experienced sexual abuse were more likely to report psychotic symptoms. Scheller-Gilkey and colleagues (in press) found that individuals with a diagnosis of schizophrenia and concurrent substance use (a risk factor for relapse) had a higher frequency of childhood traumatic events, greater PTSD symptomatology, and higher depression scores. Second, studies have demonstrated that childhood sexual abuse is linked to co-occurring hallucinations and delusions in other diagnostic populations. For example, Hammersley and colleagues (2003) have shown an association between childhood abuse and hallucinations amongst individuals diagnosed with bipolar disorder. Third, epidemiological studies have shown a link between childhood trauma and psychotic experiences. Bebbington et al., (2004) identified psychiatric disorders amongst 8580 individuals living in the UK. Compared to respondents with other psychiatric disorders the prevalence of lifetime victimisation amongst people with definite or probable psychosis was elevated. These experiences included sexual abuse, bullying, local authority care, running away from home, being a victim of assault. After controlling for the possible interrelationship between events; sexual abuse, running away from home, being in a children's home, expulsion,

homelessness and assault all remained significant predictors of psychosis. Controlling for current levels of depression, childhood sexual abuse remained the most significant and powerful risk factor for psychosis. Finally, after controlling for both depression and the inter-dependence of events, sexual abuse, being expelled from school and experiencing assault were predictors of having psychosis.

What is apparent from these data are that severe disruption in early attachment and bonding experiences increase individuals' vulnerability to developing psychosis. In a general population sample of 4045 participants, who were followed up over two years, Janssen and colleagues (2004) found that experience of childhood sexual abuse associated with psychosis. This relationship remained despite different types measurements of psychosis. In addition more frequent sexual abuse was associated with greater risk of developing psychosis and having need for care.

There is growing evidence that psychosis is experienced as a traumatic event. Eight studies have investigated the prevalence of post-traumatic stress disorder (PTSD) symptomatology following psychosis (Frame & Morrison, 2001; Kennedy et al., 2002; Meyer et al., 1999; McGorry et al., 1991; Priebe, Broker & Gunkel, 1998; Shaw, McFarlane, & Bookless, 1997; Neria et al 2002; Jackson, Knott, Skeate & Birchwood, 2004). These studies reported that between 11% and 67% of individuals meet criteria for PTSD following an acute episode of psychosis, although the prevalence of trauma related symptom clusters such as recurrent intrusive memories is considerably higher in some studies (e.g. Meyer et al., 1999).

Most studies indicated that the experience of the psychotic symptoms themselves was primarily responsible for patients' trauma (Frame & Morrison, 2001; Kennedy et al., 2002;

Meyer et al., 1999; Shaw et al., 2002); however, some studies have suggested that the methods used to treat psychosis may also be partly responsible (McGorry et al., 1991; Frame & Morrison, 2001). Although the methodology of these studies has been criticised (Morrison, Frame & Larkin, 2003) and despite the fact that the experience of an acute episode is not formally recognised as an event which fulfils DSM IV (APA, 1994) criterion A for PTSD, the findings still appear to indicate that many patients experience significant post-traumatic stress symptomatology, which arises following the treatment and experience of acute psychosis. Participants in these studies reported intrusive recollections of stressful hospitalisation events such as police involvement, or symptom based experiences including uncontrollable auditory hallucinations, persecutory paranoia, thought broadcasting and passivity phenomena. Individuals with a 'sealing over' recovery style were more likely to report fewer intrusions and greater avoidance when assessed using the Impact of Events Scale (Jackson et al., 2004). Those participants with greater levels of peri-traumatic depersonalisation derealisation and numbing also had greater levels of intrusions and avoidance (Shaw et al., 2002). Shaw et al., (1997) found that experiences representing loss of control were rated the most distressing by individuals. These "loss of control" experiences, which included enforced seclusion, experiencing the self being controlled by external forces, visual hallucinations, and thought insertion were associated with the highest levels of distress.

4.2.4 Interpersonal coping

On the evidence of the above discussion, the connection between attachment styles, interpersonal functioning and utilisation of social supports seems clear. Sarason and colleagues (1990, 1991) investigated the link between perceived social support and adult attachment styles. They noted that avoidant individuals hold representations of self and

others that make them prone to encoding and recalling instances of helpful behaviours as less supportive. In a similar vein, Blain et al., (1993) showed that higher levels of perceived support occurred among secure individuals. Ognibene and Collins (1998) also found higher support perceptions among secure individuals and significantly lower levels among fearful avoidant persons. On another dimension, social support and attachment styles also seem to be related in terms of the utilisation of available supports and the search for support.

Wallace and Vaux (1998) found that individuals with an insecure attachment representation hold a more negative support network orientation. Mikulincer et al., (1993) demonstrated that individuals with a secure attachment style use social support seeking to a greater extent than insecure ones. Mikulincer and Florian (1997) further found that attachment style also was a mediator for the impact of social support. In an experimental task they determined that conversing with a close other about the emotional and instrumental aspects of a stressful and distressing event reduced negative affect among secure individuals whereas avoidant individuals appeared to benefit only from instrumental support in the same condition and anxious/ ambivalent individuals showed increased negative affect in the condition of emotional support being offered.

On the other hand, an alternative set of possible models for the effect of social support on emotional health, particularly in the presence of stressful life events, has centred on direct effects and so called indirect or buffering effect of social supports (for reviews refer to Alloway & Bebbington, 1987; Landerman et al., 1989). In terms of a direct social support effect, low levels of social support and impoverished or absent confiding relationships are associated with poor emotional health and a significant vulnerability to mental disorders (for an overview Cohen & Willis, 1985) . The so-called 'buffering

hypothesis' of social support states that social support has a protective function in the face of stress and that in the absence of high levels of stress social support will show no relationship with mental health (e.g. Brown, 1989; Parry & Shapiro, 1989). The model of social support proposed by Brown & Harris (1978) is the principal example of a 'buffering' model and has been replicated in numerous studies, including longitudinal investigations of the impact of early negative experiences and the effect of social support over long developmental timeframes (Champion et al., 1995). The main vulnerability factor emerging from this research is the lack of a supportive network and particularly the lack of an intimate or confiding relationship.

It is known that social support from family and friends can act as a bulwark against psychosocial stressors and enhance functioning among individuals with an experience of psychosis. A number of studies demonstrated that individuals suffering from a bipolar disorder experience less positive social support and that low social support is associated with affective relapse (Romans & McPherson, 1992; Kulhara et al., 1999; Beyer et al., 2003). In two prospective studies, Johnson and colleagues (Johnson et al., 1999, 2000) found that poor social support predicted a higher number of relapses and longer time to recovery; the main mediators for relapse were higher depressive symptoms and low self-esteem. However, social support and social stress are not innovative concepts or concepts particular to psychosis, like 'parenthood' these are inherent to human sociability and associations between mental well-being and social connectedness has been stressed by early authors (e.g. Burton, 1621). In that respect we are not faced with a unitary concept but any consideration of social or developmental risk factors depends on their context and on their given operational definitions.

In an attempt to understand the processes further by which social support exerts its effects on emotional health a number of mechanisms need to be considered. In particular it is important to consider the likelihood of confounding effects in the measurement of social support and the assessment of stress. Sources of support are often also sources of stress; and as the perception of stress does not occur independently from the perception of support. Significant stressors can directly change the availability of supports or the utilisation of existing supports. Schuster et al., (1990) pointed out that most relationships involve positive and negative aspects and that close interpersonal networks are most likely to be sources of stress. The processes by which negative aspects of close relationships and networks exert their effects on the emotional health of individuals is likely to be equally complex, and negative aspects are likely to have direct and indirect effects. Some of the investigations in the negative aspects highlighted that the negative effects of social networks might outweigh the positive ones in terms of their influence on mental health (e.g. Schuster et al., 1990). It is predominantly qualitative aspects of interpersonal relationships that seem to have significant effects, such as experiences of being let down (Brown et al., 1986) and lack of reciprocity in relationships both in terms of not receiving sufficient support from close others but also significantly in terms of building up 'caring debts', the feeling of not being able to give back received support (Pearlin, 1985).

Individuals suffering from a bipolar disorder or psychosis might be especially vulnerable to the particular interactions associated with high EE, such as increased criticism or emotional involvement. It appears to be a connected characteristic that individuals suffering from bipolar disorder tend to have very small social networks which are usually also very dense in terms of the interconnectedness of network members and consist mainly of relatives and professionals (Cohen & Kochanowicz, 1989). However smaller

networks are often associated with increased satisfaction with the available support (Schwannauer, 1997).

Throughout the research literature looking at the specific effects and functions of interpersonal relationships on emotional well being, social support is conceptualised as an external and mainly structurally stable component. This can be misleading as it underestimates the dynamic and changeable nature of most interpersonal relationships and the active role that individuals play in creating and maintaining most of their significant relationships. Social support and the positive and negative effects of significant interpersonal relationships need to be understood from a developmental and dynamic perspective which will also shift the focus on the inter-relationship between stressful life events and support. In a long term follow up study, Champion (1995) found that a lack of emotional support was associated with an increased rate of negative events.

4.2.5 Individual adaptation to onset of bipolar disorder

Finally, consideration needs to be given to the individual's cognitive and emotional response to the experience of a first episode of bipolar disorder. In particular, the appraisal of the disorder has been strongly linked to depression subsequent to the abatement of acute psychotic symptoms. Firstly, Birchwood, Mason, MacMillan and Healy (1993) showed that depression following an acute episode of psychosis was associated with individuals' perception of being unable to prevent or control relapse (e.g. "I am powerless to influence or control my illness") or the fear of psychosis itself (e.g. "My illness frightens me"). Rooke and Birchwood (1998) followed up this group of patients 2.5 years later. In this group, levels of depression were persistent over time, as were appraisals of entrapment (inability to control or escape from psychosis), loss of social

role and self-blame. Individuals who were depressed felt greater entrapment and loss in relation to their psychosis. In addition there was evidence that these appraisals were consistent with participants' personal experiences of psychosis. For example, participants with depression were more likely to have experienced more compulsory admissions and loss of, or drop in employment status. Theoretical perspectives derived from evolutionary psychology as exemplified by social ranking theory (Gilbert 1992) provide theoretical framework to explain these findings. A person's perception of their social attractiveness and acceptability to others confirm their sense of rank, importance and place within their social and interpersonal environment.

Therefore life events that evoke feelings of loss (e.g. loss or disruption in important attachments or friendships) or events that threaten an individual's social ranking or importance (e.g. feeling humiliated by an episode of psychosis) are depressogenic via their impact on the lowering of perceived self-esteem and social status. In relation to people with a diagnosis of schizophrenia, these processes can be observed in two important recent studies. Birchwood, Iqbal, Chadwick and Trower (2000) found that, in a sample of 105 individuals, a proportion of 36% developed Post Psychotic Depression (PDD) without concomitant changes in positive and negative symptoms. Participants who developed PPD were more likely than their non-PPD counterparts to attribute the cause of psychosis to themselves (self-blame), perceive greater loss of autonomy and valued role, and perceive themselves as entrapped and humiliated by their illness.

In addition, individuals with and without PPD aspired to similar social and vocational roles. However, consistent with the predictions of social ranking theory, those who developed PPD saw their future status as lower. These participants also had greater insight into having a psychotic illness. Therefore, psychosis can be conceptualized as a life

event that triggers depression via awareness of its social, interpersonal and affiliative implications. Individuals, who develop depression following psychosis, appraise this life event as representing a humiliating threat to their future status, leading to the loss of valued social roles, from which escape is blocked due to actual or feared relapse, or indeed persistent symptoms.

4.3 Summary

In the above chapter key concepts were introduced that are relevant to the design and implementation of a psychological treatment for bipolar disorder. In addition to key concepts outlined in cognitive behavioural approaches in relapse prevention, mood regulation and the targeting of underlying cognitive vulnerabilities we need to consider the impact of social environment, interpersonal vulnerabilities and sensitivities, and the impact of developmental factors on any individual's susceptibility to frequent mood instability and recurrence of symptoms.

Any approach addressing complex and enduring relapse courses needs to target these interacting factors over time and within the individual's particular cultural and socioeconomic context. First we discussed the evolution of individual cognitive vulnerability through the development of negative cognitive interpersonal schemata. This has been suggested by the findings showing that early emotional instability, stress reactivity and low self esteem heighten risk for the development of bipolar disorder. Second we see the emergence of individuals' interpersonal vulnerability via their experience of core attachments, peer relationships and the successful utilisation of social supports and networks. This is suggested by the evidence for the role of early adolescent social isolation and high sensitivity to negative interpersonal environments and stressful life events. Third, we see that those who develop bipolar disorder are at heightened risk of experiencing a range of traumatic events including sexual abuse, which have the capacity to undermine core attachment experiences and positive affiliation as well as the utilisation of available supports.

We also see the importance of increased risk arising from living in an urbanised and fragmented social environment where themes of interpersonal mistrust and fragmentation may mesh with existing negative cognitive interpersonal schemata. It is in this context that bipolar disorder itself provokes major affective responses in individuals and their social environments. These affective reactions are mediated via appraisals of danger, loss, entrapment and humiliation. This may occur in the context of an already weakened platform for the regulation of affect conferred by the experience of a range of pre-existing adverse social events and/ or social isolation.

V Predictors of symptom severity and treatment outcomes in bipolar disorders – a randomised controlled treatment trial

5.1 Introduction

Manic depression is one of the most long-term recurrent mental illnesses. Despite the efforts in pharmacological management of manic depression, relapse and residual symptoms remain a major factor in the development of illness chronicity, and social and occupational disability. For individuals themselves relapse is critical in the development of secondary psychological morbidity (e.g. Birchwood *et al*, 1993; Jackson *et al*, 2000).

Up until very recently bipolar disorder has generally been understood as a biologically based disorder the treatment of which is limited to psychopharmacology. Nevertheless, longitudinal studies have suggested that even when patients are protected by state-of-the-art pharmacotherapy, about 40% relapse within one year and up to 73% over five years (Gitlin *et al*, 1995), and at least half of patients who do not relapse suffer from a high level of residual symptoms (Gitlin *et al*, 1995; Harrow *et al*, 1990); in one study 19% of individuals with bipolar disorder were found to die from suicide (Isometsa, 1993). With the growing recognition of the social, emotional and psychological costs of relapse studies are emerging to examine approaches to the detection and prevention of relapse, and to investigate more stable ways of the clinical management of this disorder. Psychosocial interventions have been shown to provide an essential adjunct to the traditional forms of treatment for bipolar disorder (e.g. Scott, 1997; Miklowitz *et al*, 1997; Frank *et al*, 1999). Most of the existing models of psychosocial intervention are family focused and

psychoeducational, or attempt to engender, on an individual basis, strategies for minimising the impact of disrupting life events and illness related behaviours. We need therefore more comprehensive theoretical and empirical approaches to understand the prognostic roles of psychosocial factors in bipolar disorder and models of psychosocial intervention that follow from these theoretical approaches.

A key aspect of management and relapse in psychotic disorders, and manic depression in particular, is the detection of so-called early warning signs that, if carefully monitored and managed, can reduce the likelihood of a relapse occurring. Birchwood *et al* (1998) point out that prodromal symptoms and early signs are subject to considerable variance in their character and timing. These individual variations in the nature and timing of early signs will act to reduce their apparent amplitude in group studies, and increase the likelihood of false positive and false negative predictions of relapse in clinical practice. Therefore, it may be more appropriate to think of early signs as an individualized configuration of symptoms. In order to improve the effectiveness of early warning sign systems and make them relevant for clinical practice a theoretical model of relapse in bipolar disorders needs to be developed. We suggest that such a model would need to incorporate negative beliefs about self and illness associated with the external and internal events, 'early warning signs', which have strong similarity with previous relapse.

Definitions currently used to capture sensitivity of early signs could well benefit from definitions more closely allied to those negative beliefs and emotional experience about illness and self, which are hypothesised to dictate relapse speed and acceleration (e.g. Gumley *et al*, 1999), rather than relying on a more closely delineated set and individual symptoms alone. This model would also need to include other significant risk and vulnerability factors that are commonly associated with relapse and negative outcomes

such as wider psycho-social factors, social support and expressed emotion, and variables of cognitive vulnerability such as perceived control over illness and meta-cognitive processes. If clinicians pay sole attention to the occurrence of generic signs and symptoms, they risk failing to capture the more holistic and generic meaning experienced by the individual. Therefore models of early signs which are more closely allied to the beliefs and psychological processes activated during early relapse may increase sensitivity and reduce the apparent variance in the nature and the timing of experiences signalling future relapse.

Bipolar disorders frequently involve repeated relapse and long hospitalisations. There is substantial evidence, that well targeted, individualised psycho-social interventions can make a considerable impact on the clinical status and social adjustment of the chronically ill patient (Scott, 1997; Miklowitz *et al*, 1997; Frank *et al*, 1999). Such interventions reduce relapse rates and significantly improve the quality of life of the patients concerned (Birchwood *et al*, 1992). We believe that so far there has been a relative neglect of research into psychosocial and psychological process variables in the onset and course of bipolar disorders, and, compared with advances in schizophrenia over the past 5-10 years, we know little about the clinically relevant theoretical models of bipolar disorders.

Social relationships and social support have been shown to have robust effects on a broad range of psychiatric and biological outcomes. Generally, on the one hand, the literature is replete with evidence that supportive relationships act as a protective factor in those vulnerable to psychiatric disorder (Champion, 1994). However, more research attention is needed to address how social support may prevent relapse and maintain good functioning in chronic patient groups. Because these chronic groups of patients make the heaviest use of professional resources, it is both desirable and necessary to

obtain a better understanding of how to facilitate more effective support for people suffering from bipolar disorder acknowledging their distinct needs. George Brown's (Brown & Harris, 1978; Brown, 1989) work on psychosocial factors pre-disposing to depression in women is perhaps the best-known representative of this strand of work. He noted that a close confiding relationship was a protective factor for women vulnerable to depression. On the other hand, Rutter and Brown earlier noted that depressive patients were particularly vulnerable to high rates of critical comments in immediate relatives. Indeed, they seemed to be more so than schizophrenia sufferers. It was, though, in this latter population that further research into the potentially adverse effects of particular aspects of social interaction took place. Vaughn and Leff's (1976) work in the 70's looking at the effects of spawned a host of studies exploring the detrimental effects of emotional over-involvement in the families of people suffering from schizophrenia or major depression. This, together with high rates of critical comments, emerged into the concept of "expressed emotion" as one of the most robust psychosocial variables predictive of relapse in schizophrenia.

It becomes clear then that the concept of social support is more than a single unitary phenomenon, whose complex interactional effects are often overlooked. Indeed it is likely that those individuals with whom we interact most closely will offer both positive and negative social interaction. Moreover, our clinical decision-making may well involve a judgement as to whether a person's condition is being affected by a lack of social support, a surfeit of negative social interaction, or a combination of the two. Franks (1992), for example in an investigation of social support in people with depressive symptoms noted a positive relationship between symptom severity and (a) high levels of criticism, (b) negative life events and (c) low levels of supportive interactions with others. However, after controlling for high criticism the association of low social support with depressive

symptoms was no longer statistically significant. This study suggests therefore that negative family interaction was more important than positive social support in this group.

Social support and Expressed Emotion both contribute to the association between psychosocial factors and the severity of psychiatric disorder; there is a significant additive effect of both measures regarding their power to explain relapse, severity of symptoms and chronicity of the disorder (Schwannauer, 1997). This model further helps to create a more detailed and valid formulation of the vulnerability-stress model for different diagnostic groups that can inform disorder specific psychosocial interventions.

There is also a considerable connection between the two psychosocial measures of social support and Expressed Emotion itself (Schwannauer, 1997). Consequently on a conceptual and methodological level social support cannot solely be looked at in respect of its positive and beneficial effect on the psychological well-being of psychiatric patients, but also has to be seen in its stressful and negative aspects. On a conceptual level this implies turning the concept of social support into a multi-axial concept including both aspects of social support and social strain. It is not, as often assumed, the sheer availability of social support, which might have a restorative effect on mental health but rather the perceived quality of the existing social links and the form of the interactions available. These negative social bonds can presumably take many forms (such as: supportive actions that misfire, indirect social stress which befalls close persons, 'hazards' presented by social relationships, rejection or demand from others, etc.) and the qualities which are conceptualised within the 'Expressed Emotion' concept present a certain proportion of these; others as well could be included in a wider concept of social support. On a more practical level, possible psycho-social interventions or preventional measures have to take into account these particular aspects of social interactions and be

tailored at one hand to the particular needs of certain client groups as well as to the different interactions of qualitatively distinct support functions.

Further there is a growing evidence base that in bipolar disorder as in depression and psychotic disorders there is a significant influence of life events on the onset of both manic and depressive affective episodes, but that the role and relevance of specific life events can change regarding nature and severity and its influence on different mood states (Johnson & Roberts, 1995; Ramana & Bebbington, 1995). It is still unclear what role psychosocial moderators of life events play in bipolar disorder, models derived from unipolar depression do not seem valid, as life events in this disorder group seem to influence social rhythms as well as cognitive vulnerability, expectations, which might vary in different mood states.

Recent advances in cognitive therapy and more generally in the emerging models of psychological interventions in schizophrenia have demonstrated that an individual's cognitive appraisal and belief systems of both symptoms and life events and social interactions play a major part in the effects of these factors on onset and course of severe psychiatric illness. Current psychological conceptualisations of positive symptoms in schizophrenia for example, hold that these symptoms contain material that is personally relevant to the individual, and that this personal relevance is critical to the understanding and cognitive interventions with these symptoms. Further that an individual's attempt to assimilate and accommodate the changes associated with severe psychiatric illness are central to the development and maintenance of symptomatology.

Sense of self and self-concepts are further mediating factors that become instrumental in an individual's appraisal of illness factors and are likely to influence the self-management

and monitoring of symptoms (e.g. Birchwood, 2000). Recent conceptualisations of the role of self-concept in both depression and bipolar disorders hypothesise that individuals suffering from mood disorders tend to experience more 'self-ambivalence', i.e. generally more positive and negative emotions about the self (e.g. Power, 1991). In bipolar disorder this model is further exaggerated in that certain emotional experiences are associated with more extremely negative or positive aspects and beliefs about the self (Power & Dalgleish, 1997). These observations about this more extreme style of organisation of the self have been demonstrated by Showers (1992), contrasting the notion of 'compartmentalisation' vs. 'integration' of the self, which she found to be correlated with self-esteem and depression scores. In a compartmentalised self-structure the individual with more positive self esteem would activate none, or very few counterbalancing negative elements and vice versa, whereas in an integrated self-structure the individual would be able to balance positive and negative aspects about the self. For individuals suffering from bipolar disorders this process is likely to compromise the adaptive regulation of emotional experiences and further the experience and development of extreme mood states.

In combination with the above-mentioned cognitive appraisal of symptoms, this study aims to examine these aspects of self esteem as important factors regarding the self-efficacy and the self-management of illness-related difficulties.

It is therefore the aim in this study to move away from a structural and solely epidemiological understanding of psychosocial, cognitive and emotional risk factors, towards a more process oriented model of perception and cognitive processing of these factors during varying stages of the disorder, and to investigate the connection of these processes with the development and course of the illness. Particularly in bipolar disorder

it can be hypothesised that the rapid changing and bidirectional nature of symptomatology and frequent switch of mood states have a profound effect on the perception and working of psychosocial, cognitive, and emotional risk factors. Recent advances in psychotic disorders further demonstrate that the development of co-morbid difficulties and 'treatment resistance' relate to high levels of trauma, negative consequences and life changes, and are mediated by changes and differences in attributions and subjective meanings;; again in bipolar disorder an important additional factor might be the interaction of these factors with changing mood states, as in other disorder groups there are marked differences between depressive and psychotic phases.

We aim to examine in detail the association between psychosocial risk factors and idiosyncratic cognitive appraisal of symptoms, illness beliefs, and emotional experience.

5.2 Aims & Hypotheses

The primary aim of this study is to examine the efficacy and effectiveness of a psychological intervention for individuals suffering from bipolar disorder. The primary outcome is quality of life, secondary outcomes are bipolar symptom severity, relapse and hospital admissions following psychological treatment compared with treatment as usual.

The study further aims to investigate the relationship between psychosocial, cognitive and emotional risk factors and the severity and recurrence of bipolar disorder. It also aims to evaluate a model of psychological therapy that was developed specifically with the complexity of presentation and underlying psychological processes in bipolar disorder in mind.

The psychological treatment developed for this trial was offered in two modalities, individual and group intervention. Differences in outcomes for the two treatment modalities will be explored.

5.2.1 Hypotheses

1. A) There will be a positive change in measures of quality of life, severity of symptoms and indicators of relapse in individuals with bipolar disorder following psychological intervention and treatment as usual compared with treatment as usual alone.

B) There will be no significant difference in the treatment effects between the two treatment modalities, group and individual treatment, in quality of life, severity of symptoms and indicators of relapse.
2. Psychological intervention will effect positive changes in psychosocial, cognitive and emotional factors.
3. Changes in psychosocial, cognitive and emotional factors will be predictive of improved quality of life, reduced severity of symptoms and reduced indicators of relapse.

A) There will be specific and differentiating interacting and mediating effects of cognitive, interpersonal and psychosocial variables relating to levels of depression and mania at baseline.

B) There will be a full mediational effect of cognitive, interpersonal and psychosocial variables on outcome for levels of depression and mania.

5.3 Study Design

The design for this study was a pragmatic clinical trial with random allocation to treatment and waiting list control and prospective follow-up at 6 months and 18-months following end of treatment. The study compared treatment as usual alone (TAU alone) over a period of six months while waiting for treatment, with Integrated Cognitive and Interpersonal Therapy plus TAU (CIT + TAU). Following allocation to the treatment group, patients had the choice of psychological treatment in either group or individual treatment format. The study evaluation followed a mixed design of a between subject analysis between the two treatment groups and a longitudinal within subject analysis for the group following psychological treatment.

The element of choice within the treatment condition, for participants to decide on which treatment modality they want to participate in following randomisation to treatment or after the waiting period, was favoured by users in the project steering group and by the Bipolar Fellowship Scotland (BFS) who supported the trial. It was felt that as part of any service provision users of that service should have a free choice of available treatments. Further the BFS had experience of developing peer support groups throughout Scotland and felt that engagement and adherence would be significantly improved if participants had a choice and opted into a group format rather than being allocated to a particular treatment modality. We also know that interactive group psychotherapy models tend to be more effective if participants are well socialised into the group therapy model and if their personal treatment goals can be linked to the format and structure of group treatment (Yalom, 1995).

The treatment trial was conducted between 2000 and 2003, followed by a follow up period of another 18 months. Patients with a confirmed life time diagnosis of bipolar disorder from the Adult Mental Health Division in Lothian were recruited into the study. All Consultant Psychiatrists and Community Mental Health Teams within the Division were approached and informed about the trial and asked to make the opportunity known to all patients on their caseload that fitted the criteria for inclusion in the study. The locality area for the service includes a population of approximately 720,000 encompassing rural, suburban, and metropolitan areas. Inclusion required that patients fulfilled DSM-IV (American Psychiatric Association, 1994) criteria for bipolar I disorder. The diagnosis was confirmed by the referring psychiatrist and independently by the research assistants employed in the project using the Structured Clinical Interview for DSM-IV (First et al., 1994).

Individuals were excluded from the study if they were a non-English speaker, had organic brain disorder, presence of significant learning disability, or were in the receipt of ECT or a concurrent psychotherapy intervention outside the study.

Over the first three years the study was supported by the Bipolar Fellowship Scotland (BFS), then: Manic Depression Fellowship (Scotland). Together with members of the research team, ordinary members and members of the board of the BFS a steering group was constituted at the start of the project which met regularly throughout the first two years of the trial. The steering group offered advice and guidance on the design and implementation of the treatment and the treatment trial and supported important processes regarding recruitment and consent. The active involvement of the Bipolar Fellowship Scotland greatly aided recruitment and retention of individuals who participated in the trial. The background to this is a long history of BFS lobbying for

improved access to psychological treatments and their development of user led self-management groups in which the trial was promoted.

5.3.1 Power

The estimated sample size was calculated on the basis that a sample of 110 participants (55 per group), would have 80% power to detect at $p < 0.05$ a reduction in symptoms and relapse rates from 40% in the TAU alone group to 20% in the CIT and TAU group. Power calculations were carried out using the software Sample Power 2.0 (SPSS Corp., 2000), and where necessary calculated using the method suggested by Bortz and Doering (1995). The calculations showed that for a two group comparison comparing treatment with non-treatment groups and a comparison of the two treatment conditions, TAU and TAU + CIT, at a power of 80% and with alpha at 0.05 a minimum number of $N = 45$ would be necessary in the total treatment and TAU groups. These calculations are assuming a medium effect size based on reported effects of psychological intervention in bipolar and comparable treatment groups (e.g. Lam et al., 2000). In order to enable a systematic comparison of all outcome indicators between the two treatment conditions an estimated number of $N=45$ was necessary in each treatment condition using the same estimates of power and effect size. In order to equalise numbers in the TAU and treatment groups overall and to allow a systematic comparison of the two treatment conditions we aimed to recruit a total of 180 participants with a confirmed diagnosis of bipolar disorder, aged between 15 and 65 randomly allocated to the treatment and waiting list control conditions.

All patients in the treatment group were assessed at intake, mid-treatment, end-treatment and at 6 month and 18 month follow-up. Psychosocial measures included

quality of life, life events, social support and expressed emotion; psychological measures included self esteem, beliefs about illness, and meta-cognitions. Relevant symptom measures included measures of depression, mania, and mixed episodes. In addition to these self-report measures, all subjects were assessed utilising observer rated measures of severity of symptoms and a case note review including documented relapse, and service use before, during and following the intervention. Individuals' perception of the therapeutic process was assessed at follow-up. The assessments were carried out by research assistants who were blinded to the allocation at baseline and at start of treatment.

5.4 Ethical Approval

The Regional Research Ethics Committee for Lothian approved the study protocol and design as well as the patient information sheet and consent form employed in the study. Patients were made aware that they were randomly assigned to psychological treatment or a waiting list for the same psychological treatment and that their psychiatrist and GP would be informed of their progress and outcome at the end of treatment.

Originally randomisation to a treatment as usual condition for the whole study and follow-up period was planned for the trial. The potential clinical implications for such design were raised by the research ethics committee in an area where no comparable service existed for individuals with bipolar disorder and consequently converted into a waiting list control condition.

5.5 Psychological Treatment

The psychological treatment implemented in the trial consisted of key elements of relapse prevention, cognitive behavioural therapy for bipolar disorder, and essential elements of interpersonal psychotherapy approach incorporating essential components of the psychological treatments reviewed above. The treatment offered was modularised in 8 distinct modules, consisting of: Psychoeducation, individualised profile of early warning signs, developmentally based individual formulation, behavioural strategies for coping with mania and depression, formulation based cognitive strategies targeting underlying beliefs and assumptions that increase vulnerability to mood dysregulation, affect regulation techniques, social network analysis, and the identification of interpersonal patterns and strategies. For each participant in the individual or group modality a treatment plan was formulated and certain modules were emphasised depending on the individual's formulation, needs and negotiated goals. For a detailed description of elements and principals of the integrated treatment please refer to Gumley and Schwannauer (2006).

The group therapy format consisted of 20 weekly sessions of one and a half hour length and the individual treatment consisted of 18 to 20 weekly one hour long sessions. The groups were carried out in group treatment facilities on the premises of the Royal Edinburgh Hospital. Groups usually consisted of 8 participants (± 2) and were led by one therapist, usually joined by a training grade psychiatrist or clinical psychologist in training. Prior to the commencement of the group all participants received two individual sessions during which an individualised formulation was formed and shared with the individual and specific individual treatment goals were formulated in view of the group format of the treatment. This included the orientation to the group outline and session format as well as some of the particular issues of group treatment, such as confidentiality, listening and

learning from the experience of others as well as dealing with upset and other difficult emotions within the group setting. Particular emphasis was placed that all participants had an idea how the group could help them to achieve their personal goals for a psychological treatment.

During the first year of the trial two groups were carried out in parallel. The content of the group treatment was informed by the same modules as the individual treatment. Psychoeducation, individualised profile of early warning signs, developmentally based individual formulation, behavioural strategies for mania and depression, formulation based cognitive strategies, social network analysis and the identification of interpersonal patterns and strategies.

An added element of the group therapy modality was the interactive group format. Participants were encouraged to set group tasks from session to session in line with the modules discussed, e.g. early signs monitoring, or cognitive strategies targeting particular assumptions and beliefs. These were then discussed in the following group sessions and participants benefit directly from other participants' experiences and reflections. We further found that the interpersonal component of the group format strengthened the validity of particular interventions, such as behavioural interventions aimed at stabilising individual mood patterns, as these were reinforced by the group and shared experiences. Other aspects that strengthened the effectiveness of the group treatments were universality, normalising of bipolar experiences and extreme mood states, social learning, shared experiences and understanding and reduced shame and hesitation in discussing openly experiences related to past bipolar episodes. The group further was able to instil optimism and hope through the sharing of difficult experiences and individual participants' sharing of recovery and mood stabilisation. An important aspect of the group was its

function to facilitate interpersonal learning and to provide corrective emotional experiences.

On reflection it seems that the benefits for the participants in the individual treatment format were the flexibility of the sessions and the strongly individualised treatment goals. The added advantages of the group treatment included the normalising and destigmatising effect of shared experience, and the shared learning from other participants' past experiences of successful application and modification of the psychological intervention techniques.

All treatments were delivered by two therapists, Matthias Schwannauer, Chartered Clinical Psychologist and IPT/ CBT therapist, and Sharon Fegan, Occupational Therapist and IPT/ CBT Therapist. Both therapists were employed on the trial on a full time basis and carried out all individual and group treatments with trial participants. Both therapists had previous formal training in cognitive behavioural therapy and interpersonal therapy prior to the trial as well as several years of clinical experience with psychiatric patients suffering from severe and enduring mental health difficulties.

5.6 Sample

As outlined above, participants for the trial were recruited from the Adult Mental Health Services in Lothian, comprising of five Community Mental Health Teams (CMHT); four teams in Edinburgh and one community mental health team in Midlothian. According to the Patient Information and Management System (PIMS), 648 patients with a diagnosis of bipolar I disorder were registered with the participating teams over the course of 2000

to 2003. 258 individuals were identified by the referring teams as fitting the inclusion and exclusion criteria and who were willing to participate for inclusion in the trial. Of those, 36 did not meet diagnostic criteria according to the SCID (First et al., 1994), 16 did not attend for the original assessment appointments for the trial and 4 were outside the specified age range. 212 individuals completed the baseline assessments and were randomised into two experimental conditions, psychological treatment plus TAU vs TAU and delayed psychological treatment, with 106 participants each group. The assessments were carried out by research assistants. All participants provided written informed consent.

Randomisation took place following a computer generated number sequence, to which the research assistants, therapists and referrers were blind. Following the allocation, 9 participants were excluded from the trial; of those, 3 withdrew consent and 6 were excluded by RMO following a change in diagnosis. 1 participant randomised to the TAU group committed suicide following the baseline assessment. From the 202 individuals who entered the study 9 did not commence treatment following the six month wait period. There were no significant differences in the clinical or sociodemographic characteristics of those who dropped out at this stage and those who commenced treatment.

As illustrated in Figure 5.1 a number of participants withdrew from treatment before a minimum of 8 treatment sessions or did not complete all follow up assessments. Altogether 174 individuals or 86% of those who were randomised completed a course of treatment (8 or more sessions), 134 participants or 67% completed the first follow up assessments at 6 months following completion of treatment, and 108 participants or 54% completed the second follow up assessments at 18 months following the end of

treatment. The follow up assessments were carried out by research assistants employed on the trial.

In addition to the assessments carried out directly with the participants, service related data from individual medical records and the local Patient Information and Management System (PIMS) was available for 188 or 97% of participants. These data were collected by the research assistants during the follow up period of the trial and after participants had completed their individual follow up assessments. These data comprise details regarding hospital admissions and length of any inpatient stays, routine and emergency appointments and frequency of follow up by psychiatry and case managers. This information was collected for the 2 years prior to their participation in the trial, the time period during which they participated in the trial and for the 18 months following their completion of the trial or following their withdrawal from the trial.

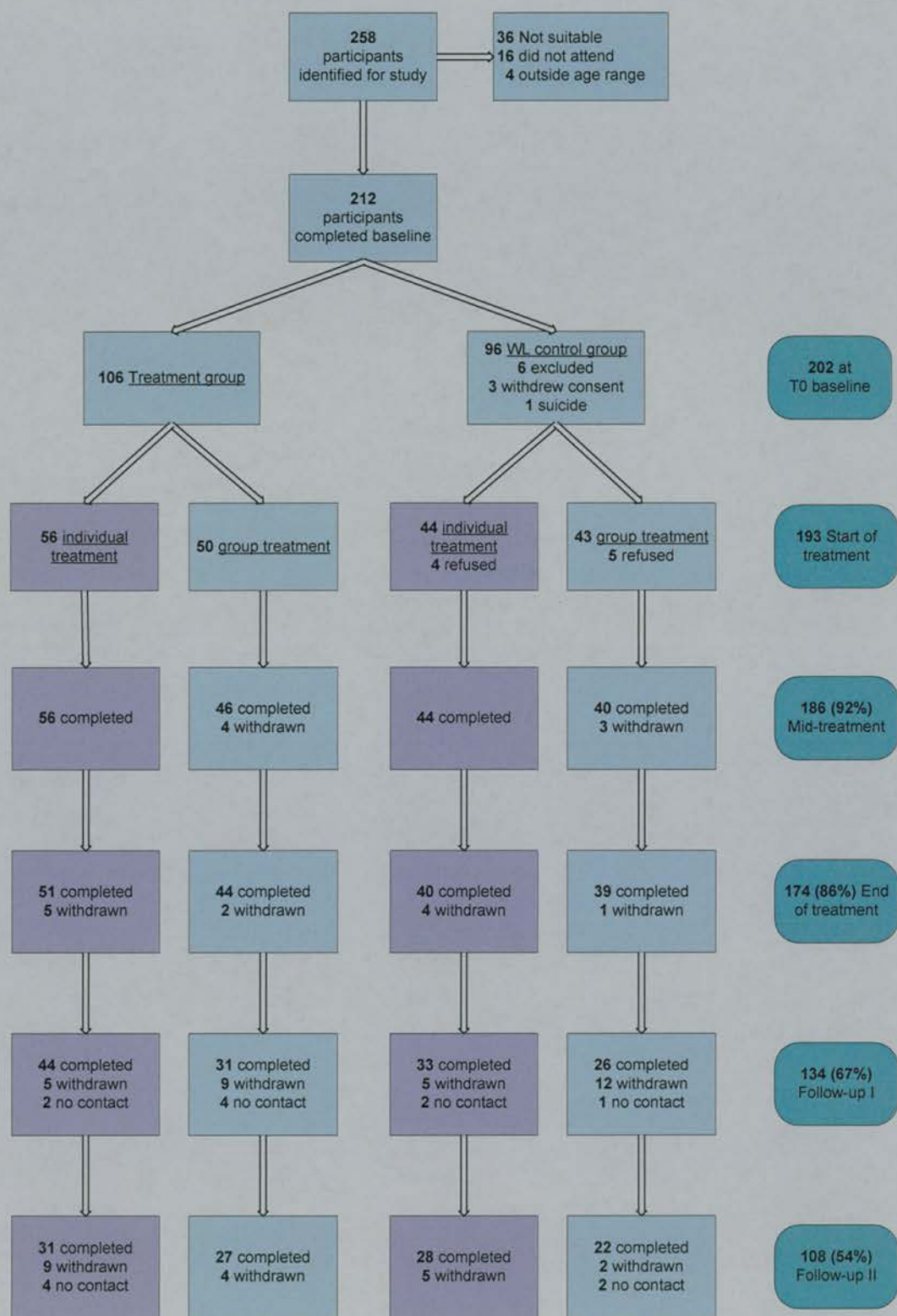


Figure 5.1: Participant Flow

5.7 Methods

5.7.1 Assessment & Measures

All individuals who entered the trial were assessed with a range of observer rated and self report measures at entry into the study, start of psychological treatment, mid-treatment (after 8 sessions in the individual treatment and after 10 sessions for the group treatment), at the end of treatment and at two follow up points (six and eighteen months after the end of treatment). The assessments were carried out in two parts and over 2 to 3 individual sessions by graduate research assistants. These consisted of two interviews of 60 minutes and a pack of self report measures that participants completed at home at their own pace over the course of one week. Following this the research assistants would go through the self report measures with the participants answering any questions and ensuring their completion. All research assistants received the appropriate training and completed inter-rater reliability checks for the observer rated measures.

The research assistants were employed in the trial and were blind to the treatment condition and to the random allocation of participants to treatment or treatment as usual at the time of the baseline assessments. The assessments were arranged following referral to the project and participants were informed of their allocation to treatment or delayed treatment following the completion of the assessments. It is not assumed that blindness to treatment condition could be completely maintained in the follow up interviews; the research assistants did not know at that point whether a particular participant was part of the TAU or the TAU + CIT group but as part of the interview participants may have mentioned a waiting time between their first contact with the project and their commencement of treatment.

5.7.1.1 Expanded Brief Psychiatric Rating Scale

The BPRS-E (Lukoff et al., 1986) is an updated, standardised and expanded version of the Brief Psychiatric Rating Scale (Overall et al., 1962). The BPRS is a structured interview centred on significant behaviour traits, which captures the nature and current degree of psychological disorders. The BPRS comprised originally 16 categories of symptoms that were assessed by a clinical person according to a seven point Likert scale, with the scale increasing according to the degree of symptoms. Items included in these scales are those to do with the most important psychological disorders, which are essential in assessing the clinical status of a psychotic patient (e.g. depressed mood, positive symptoms, delusions, grandiosity, unusual constructs, conceptual disorganisation).

In order to tailor the interview to outpatients, Lukoff and colleagues (1986) added extra items and created a standardised assessment key of all items for this group of patients. Thus, three new scales were added that ought to depict the possible relapse of psychotic patients and five additional items linked with negative symptoms, deficient behaviour or characteristics that have been shown to be particularly important for the rehabilitation of psychotic patients. Moreover a new standardised interview manual was introduced that should make uniform assessments easier. The BPRS-E can be broken down into six main dimensions (anxiety, depression, anergia, thought disturbance, activation, psychosis and relapse), four additional superior scales (verbal response, behaviour, positive symptoms and negative symptoms) and one overriding total value of global psychopathology.

A validation study by the authors showed an inter-rater reliability from 7 independent clinicians on 17 psychotic patients of $r = 0.81$. An independent evaluation of the expanded and standardised BPRS-E by Hafkensheid et al. (1991) with 162 Scandinavian psychiatric patients produced an internal consistency according to Cronbach - α from $\alpha =$

0.61 to $\alpha = 0.75$ for the individual dimensions that can be assessed as average-to-good. Moreover it demonstrated a good inter-rater reliability with $r = 0.75$ and a factor-analytical confirmation of the multi-dimensional structure of the BPRS-E.

The range of indicators of internal consistency for the subscales of BPRS in the trial was between Cronbach – $\alpha = .551$ to $\alpha = .585$.

5.7.1.2 Positive and Negative Symptoms Scale

The Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987) is a 30-item observer rated scale. Each item is rated on a severity scale ranging from 1 (absence of psychopathology) to 7 (extremely severe). The sum of the first seven items constitutes the positive scale score (e.g. delusions, conceptual disorganisation, hallucinatory behaviour, excitement, grandiosity, suspiciousness/ persecution). The sum of items 8 to 14 constitutes the negative scale score (e.g. blunted affect, emotional withdrawal). The sum of items 15 to 30 constitute the global psychopathology scale score (e.g. somatic concern, anxiety).

The range of indicators of internal consistency for the subscales of the PANSS in the trial was between Cronbach – $\alpha = .515$ to $\alpha = .854$.

5.7.1.3 Observer rated assessment of symptom severity

At the time when the design of the trial was put together there was no apparent consensus in the literature which measures of severity of symptomatic distress were best applicable to bipolar disorder. It was therefore decided to combine the two standard interview based observer rated rating tools of psychiatric symptoms, the BPRS and the PANSS into one integrated interview schedule, thus achieving scores on all subscales.

This interview schedule is attached in Appendix B. The raters were trained using interviews with patients, either face to face or on video.

5.7.1.4 Beck Depression Inventory

The Beck Depression Inventory-II (BDI-II; Beck, Steer & Brown, 1996) is a 21-item self-report instrument designed to assess the existence and severity of current depressive symptoms. Respondents are asked to rate themselves for the *past week, including today* in order to capture a more persistent 'trait' rather than just present 'state'. Items are scored on a four-point scale ranging from 0-3. The items are then summed to give a single score ranging from 0-63, giving an estimate of the overall severity of depression.

The BDI-II is an upgraded version of the Beck Depression Inventory-IA (BDI-IA; Beck & Steer, 1993), which is in turn an amended version of the original Beck Depression Inventory (BDI-I; Beck, Ward, Mendelson, Mock & Erbaugh, 1961). The current BDI-II was specifically constructed in accordance with amendments to DSM-III-R/DSM-IV criteria. New items addressing agitation, concentration difficulty and worthlessness were added and most other items were reworded in the upgrade. Other items in the BDI-II reflect a range of cognitive, affective, somatic and vegetative symptoms in order to encompass many individual characteristics of depression.

The BDI-II has been described by Beck, Steer & Brown (1996) as possessing adequate reliability and validity. It has high 1-week test-retest reliability ($r=.93$, $p<.001$) and its earlier version is positively correlated with the Hamilton Psychiatric Rating Scale for Depression (Riskind, Beck, Brown & Streer, 1987; $r = .71$).

The BDI-II has high internal consistency with a high published coefficient alpha (.91) (Beck, Steer, Ball & Ranieri, 1996). All 21 of the corrected item-total correlations were significant beyond the .001 level after Bonferroni adjustment. The same researchers also found it to have nonsignificant correlations with background characteristics of sex, ethnicity, and age.

The Cronbach – α coefficient for the BDI-II in the trial was $\alpha = .942$.

5.7.1.5 Bipolar Longitudinal Investigation of Problems Scale

The Bipolar Longitudinal Investigation of Problems (BLIP; Kavanagh, Schwannauer, Goodwin & Power, *in press*) scale was constructed as a self-report measure, though it is also designed to be used as a structured interview when necessary. Items were included that covered the following topics: Physical Activity, Verbal Activity, Thought Processes, Voice Level, Mood, Self-Esteem, Social Contact, Sleep, Sexual Interest, Eating Habits, Weight Change, Meaning in Life, Anxiety, Feelings of Pressure, Passage of Time, Future Plans, Pain Sensitivity, and Work Capacity. Each item was split into “depressive” and “manic” aspects and centred around a normative value of zero. For example, the respondent would indicate “0” if sleep level was “normal” or indicate which of a set of descriptors of increasing or decreasing need for sleep was characteristic. Each of these poles had four descriptions of increasing severity (1-4) for both the manic version of the item and the depressed version of the item. It is clear in the scale instructions that individuals could endorse *one or more* of the manic and depressive symptoms because both types of symptoms might apply at different times during an episode, for example, feeling more sociable at some points, but less sociable at other times.

In addition, the scale included a series of 19 “individual characteristics” that had been highlighted in individual clinical work, or in focus group work, but which did not fit the “bivariate” structure for the first set of items, and which only applied to a sub-group of respondents. Examples in this category included Smoking, Recreational Drugs, Spending, and Jet Lag, which are clearly important for some individuals, but which obviously are not characteristic for all.

The scale was evaluated in two samples of individuals suffering from bipolar disorder: a) a clinical sample recruited from Adult Mental Health Services in Lothian and b) a sample of community based individuals with bipolar disorder through the Manic Depression Fellowship (Scotland).

One of the questions that the novel format of this scale allows to be asked is the extent to which mixed state responses are endorsed on both an *intra-item* basis and on an *inter-item* basis. That is, an individual might respond that at times during an episode he or she felt more anxious than usual, but that at other times felt less anxious than usual, which would demonstrate *intra-item* mixed state. However, respondents could also indicate *inter-item* mixed state if they endorsed the “depressive” characteristics of one item (e.g. thought slowing) but the “manic” characteristics of another (e.g. elated mood).

Internal consistency analyses of the total 18 items were carried out using Cronbach alpha. Analysis of just the manic item scores gave a Cronbach alpha value of 0.90. The reliability analysis was repeated using the manic plus depression total scores for each item. This analysis gave a Cronbach alpha value of 0.95, with a range of corrected-item-total-scale correlations from 0.54 to 0.83. The inclusion of the summed mania and

depression scores therefore improved the scale reliability characteristics with all items now contributing substantially to the overall scale score.

The Cronbach – α coefficient for the BLIP in the trial was $\alpha = .932$.

5.7.1.6 Altman Mania Scale

The Altman Self-Rating Mania Scale (ASRM; Altman, Hedeker, Peterson & Davis, 1997) is a brief scale used to measure the presence and severity of manic symptoms by self-report. The scale consists of 5 items scored from 0 (absent) to 4 (present to a severe degree) based on increasing severity. The time for rating symptoms is generally during the most recent one week period.

The scale was constructed from a draft of 11 items, with each item representing on the of the DSM-IV defined major symptoms of mania. An additional 3 items were added for assessment of psychotic symptoms. From the original group of 14 items, items were deleted if they failed to discriminate between manic and non-manic patients. Principle component analysis revealed 3 factors accounting for over 50% of the total variance thus 3 subscales were indicated. However MANCOVA showed that subscales 2 and 3 did not discriminate manic from non-manic patients. These were subsequently dropped leaving the 5 items from subscale 1 in the final scale: elevated mood, increased self-esteem, less need for sleep, pressured speech, and psychomotor agitation.

The ASRM has good internal consistency with a published Cronbach alpha of .79. Test-retest reliability was significant with a Pearson correlation coefficient between 2 baseline ratings of $r = .86$ ($p < .001$). Concurrent validity was assessed and scores were significantly correlated with two other measures of mania: Mania Rating Scale (MRS; Young, Biggs &

Meyer, 1978) total score, $r = .718$ ($p < .001$); Clinician-Administered Rating Scale for Mania (CARS-M; Altman, Hedeker, Janicak, Paterson & Davis, 1994) mania subscale score, $r = .766$ ($p < .001$).

The Cronbach – α coefficient for the ASRM in the trial was $\alpha = .803$.

5.7.1.7 Bech-Rafaelsen Mania Scale

The Bech-Rafaelsen Mania Scale (BRMS; Bech, Bolwig, Kramp & Rafaelsen, 1979) is an 11-item scale used to assess the presence and severity of mania. Each item is defined on a five-point scale of 0 to 4 and the items are summed to give a total score ranging from 0-44.

In the construction of the scale, items were chosen for their relevance to manic behaviour as a counterpart to the Hamilton Rating Scale for Depression (HRS; Hamilton, 1960).

The Cronbach – α coefficient for the BRMS in the trial was $\alpha = .754$.

5.7.1.8 Quality of Life

The World Health Organization Quality of Life-BREF (WHOQOL-BREF; World Health Organisation, 1998) was developed to assesses “individuals’ perceptions of their position in life in the context of the culture and value systems in which they live and in relation to their personal goals, expectations, standards and concerns” (WHOQOL Group, 1998). The WHOQOL was developed to assess quality of life in both developed and developing countries around the world. Focus groups were held in 14 different countries to explore the concept of quality of life and its components. Consensus was reached about what

aspects of life were considered important and 6 domains emerged: physical, psychological, level of independence, social relationships, environment, and spirituality. Within these 6 domains 24 specific facets and one general facet relating to quality of life were identified. Each facet includes 4 questions, thus the scale yields a multi-dimensional profile of scores across domains and sub-domains of quality of life.

The full version of the scale is a 100-question assessment allowing a detailed evaluation of quality of life. The WHOQOL-BREF used in this study is an abbreviated 26-item version derived from the original which is more convenient to use. The 26 items measure four broad domains of physical health (e.g. pain and discomfort, sleep and rest, mobility), psychological health (e.g. positive feelings, self-esteem, spirituality), social relationships (e.g. personal relationships, social support, sexual activity) and environment (e.g. freedom, financial resources, participation in leisure activities), plus one facet on overall quality of life and general health. The WHOQOL-BREF was initially developed by selecting the most general question from each of the 24 facets relating to quality of life in addition to two items from overall quality of life and health. These 26 items were examined by a panel and some items were substituted or removed.

Confirmatory Factor Analysis of the four-domain structure was performed using three data sets and acceptable CFI was achieved in two (CFI= .906 and .903). In the third data set, CFI increased from .87 to .901 after some minor alterations to the scale. Domain scores of the WHOQOL-BREF were also highly correlated with domain scores based on the WHOQOL-100 (.89 to .95). Cronbach alpha values for each of the domain scores ranged from .66 to .84 demonstrating good internal consistency. Finally the WHOQOL-BREF was shown to be comparable to the WHOQOL-100 in discriminating between ill and well groups, with significant differences in all domains.

The Cronbach – α coefficient for the domains of the WHOQOL-BREF in the trial was between $\alpha = .695$ and $\alpha = .863$.

5.7.1.9 Personal Beliefs about Illness

The negative appraisals of their diagnosed disorder were assessed using the Personal Beliefs about Illness Questionnaire (PBIQ; Birchwood et al., 1993). The PBIQ is comprised of 16-items rated on a four-point scale and assesses individuals' beliefs in five domains (Rooske & Birchwood, 1998): loss of autonomy and valued social role; humiliation and loss of rank arising from a belief in social segregation of those with mental illness; shame; attribution of behaviour during illness experience to self or to psychosis; and entrapment in or an inability to control psychotic experience. The scale has been demonstrated to have good reliability and validity in a range of clinical samples of individuals suffering from psychosis.

The Cronbach – α coefficient for the subscales of the PBIQ in the trial was between $\alpha = .591$ and $\alpha = .817$.

5.7.1.10 Metacognitions Questionnaire

The Metacognitions Questionnaire (MCQ; Cartwright-Hatton & Wells, 1997) is a self-report assessment which measures individual differences in several domains of metacognitions. The questionnaire uses 65 items to generate scores for the following five sub-scales: Positive beliefs about worry (typical items include 'Worrying helps me to get things sorted out in my mind' and 'Worrying helps me cope'); Negative beliefs about the controllability of thoughts and corresponding danger (typical items include 'Worrying is dangerous for me' and 'I cannot ignore my worrying thoughts'); Cognitive confidence

(typical items include 'I have a poor memory' and 'I have difficulty knowing if I have actually done something, or just imagined it'); Negative beliefs about thoughts in general, including responsibility, punishment and superstition (typical items include 'Not being able to control my thoughts is a sign of weakness' and 'If I did not control a worrying thought, and then it happened, it would be my fault'); Cognitive self-consciousness (typical items include 'I think a lot about my thoughts' and 'I pay close attention to the way my mind works'). Items are scored from 1 to 4, whereby 1='do not agree', 2='agree slightly', 3='agree moderately', and 4='agree very much'.

The scale was originally constructed from a semi-structured interview with undergraduate students and transcripts of cognitive therapy with anxiety outpatients. Analysis of these produced 8 dimensions of meta-cognitions and 94 items representing these dimensions were chosen. Following factor extraction and factor analysis, redundant items were discarded and novel items generated. The scale was revised twice to consist of 79 items and 6 domains then finally 65 items under the 5 domains described above (Cartwright-Hatton & Wells, 1997).

The MCQ has shown good psychometric properties on a range of indices of reliability and validity. Factor analyses demonstrated that the questionnaire measures five empirically distinct categories of meta-cognition. The reliability of the five factor structure was described as relatively stable when the final version of the assessment was administered to a new sample of 243 students. The subscale scores also showed good stability over time. Internal consistency of all five subscales was good with scores ranging from .72 to .89. Four of the five subscales demonstrated significant discriminant validity when the questionnaire was administered to a clinical sample.

The Cronbach – α coefficient for the subscales of the MCQ in the trial was between $\alpha = .808$ and $\alpha = .966$.

5.7.1.11 Dysfunctional Attitudes Scale

The Dysfunctional Attitude Scale (DAS; Weissman, 1979; Weissman & Beck, 1978) was developed to measure cognitive vulnerability to depression, that is, the strength of underlying tacit beliefs an individual holds which are hypothesized to be activated by congruent stressors to produce negative affect. The DAS was originally conceptualized as a global measure of vulnerability to depression. It was designed to test predictions that people with depression subscribe to more extreme beliefs and assumptions by which they organise their lives, based on Beck's cognitive theory of depression (Beck, Rush, Show & Emery, 1979). Recently, Beck and his colleagues (Beck, Brown, Steer, & Weissman, 1991) sought to develop more specific measures of cognitive vulnerability from the DAS. Using data from pretreatment evaluations of over 2,000 outpatients, they performed an exploratory factor analysis (EFA) of the long (100-item) form of the DAS. Sixty-six of the original 100 items were retained. They found nine factors: (1) Vulnerability, (2) Need for Approval, (3) Success-Perfectionism, (4) Need to Please Others, (5) Imperatives, (6) Need to Impress, (7) Avoidance of Appearing Weak, (8) Control Over Emotions, and (9) Disapproval-Dependence. They proposed that these factors be used to address research questions which have arisen from Beck's more recent (1987) statement of his theory, which asserted that particular dysfunctional beliefs will interact with specific aspects of an individual's personality and with particular stressors. Power et al. (1994), using a sample composed of formerly depressed patients, their relatives, and general practitioner patients, developed a short form of the DAS which consisted of three subscales. They selected 24 items which appeared to measure three types of cognitive vulnerability: Achievement, Dependency, and Self-Control.

Confirmatory factor analysis (CFA) of the three-factor model found that most of the items did indeed load on the hypothesized factors.

Power et al. (1994) showed that the DAS-24 had good reliability and validity, and a close relationship with the original 100-item version. Analysis of the internal consistencies of the subscales gave acceptable Cronbach alpha values of .847, .737, and .681 for the Achievement, Dependency, and Self-Control scales respectively. Pearson r intercorrelations for the subscales were all highly significant. Discriminant validity was demonstrated on the total score and all three subscales when scores from depressed patients and their relatives were compared with GP centre controls.

The Cronbach – α coefficient for the three dimensions of the DAS-24 in the trial was between $\alpha = .568$ and $\alpha = .905$.

5.7.1.12 Rosenberg Self Esteem Scale

The Rosenberg Self-Esteem Scale (RSES; Rosenberg, 1965) was selected as a measure of negative appraisals of self. The RSES is a ten-item self-report measure of self-esteem. The scale was originally developed as a measure to assess self-esteem in adolescents but has been widely used in adult populations. Rosenberg (1965) proposed a complex scoring system, which was simplified by Corcoran & Fischer, (1987). Items are in statement form, and respondents are asked to rate their agreement on a four point Guttman Scale (strongly agree to strongly disagree). The RSES gives a score range of 4 to 40, with higher scores indicating lower self-esteem.

Originally the scale was conceptualised at a single-factor scale with 5 of the items reverse worded and total scores ranging along a continuum of low to high self-esteem. However

since then researchers have suggested a 2-factor structure to the scale comprising positive and negative images of the self (e.g. Kaplan & Pokorny, 1969; Bachman & O'Malley, 1986; Goldsmith, 1986; Owens, 1993; Sheasby, Barlow, Cullen & Wright, 2000). Several researchers have proposed that the two-factor structure is an artefact of item-wording (see Greenberger et al., 2003 for further details).

The Cronbach – α coefficient for the RSES in the trial was $\alpha = .914$.

5.7.1.13 Significant Others Scale (SOS)

The Significant Others Scale (SOS; Power & Champion, 1995) pick out the two main areas of operation of social support: “emotional support” and “practical support” for the six most important people in the respective social network. These six significant persons should, where possible, come from different areas of the social network: family, friends, neighbours, colleagues etc, whereupon the respondent can in theory choose with persons are important to him or her.

Each of the persons named are assessed by the respondent regarding the level of perceived support received and the level of ideal or desired support. The assessment of support is conducted using a seven point Likert scale from 1 (never) to 7 (always). Results show the measure of current and ideal degrees of social support that can be ascertained for each person or as a total value. Similarly, the difference between the current and ideal values as well as the perceived and desired support can be determined. This measure of discrepancy gives an appraisal of the satisfaction with the available social support.

Power et al. (1988) report in a validation study a test-retest reliability between $r_{tt}=0.73$ and $r_{tt}=0.83$, which is thus considered to be good. A construct validation by means of a factor analysis produced three factors: “emotional support”, “practical support” and “social companionship”. The authors however expounded the point of view that the factor “social companionship” can be seen as a part of “practical support” and that the data generally supports the difference between “current – ideal” and “emotional – practical”. Checks on the validity of the criteria were carried out in that three independent groups of depressed people, one non-depressive group of “psychiatric cases” and a symptom-free group were picked out and compared with regards to SOS values. The signification differences of the SOS and GHQ values between the groups of depressed people and the remaining groups support the fact that one can discriminate between the groups with the SOS, as expected.

The Cronbach – α coefficient for the dimensions of the SOS in the trial was between $\alpha = .795$ and $\alpha = .960$.

5.7.1.14 Emotional Involvement and Criticism Scale (FEICS)

The Family Emotional Involvement and Criticism Scale (FEICS; Shields et al., 1992) comprises the perceived assessment of two factors from the original concept of EE, from a recipient’s perspective.

The questionnaire covers 23 items, which can be summarised according to two factors, perceived criticism (9 items) and intensity of emotional involvement (14 items). Both of these factors are analogous with the main EE components of critical comments and emotional over-involvement that were found by the Camberwell Family Interview, CFI (Brown et al., 1972). The items consist of statements that describe the behaviour of

family members or other attachment figures. The respondents assess the frequency of the behaviour described by means of a Likert scale with 5 levels, from 1 (almost never) to 5 (almost always).

The internal consistency, defined by Cronbach - α , is all in all to be judged as good, with $\alpha = 0.83$ for the perceived criticism subscale and $\alpha = 0.76$ for the emotional over-involvement subscale. This batch of 23 items could confirm a confirmatory factors analysis with regards to both factors (Shields et al., 1994).

The Cronbach - α coefficient for the two factors of the FEICS in the trial was $\alpha = .648$ and $\alpha = .753$.

5.7.1.15 Life Events and Difficulties Schedule

Brief version (65 items) (Champion et al., 1995) of the *Bedford College Life Events and Difficulties Schedule* (LEDS; Brown & Harris, 1978). The LEDS interview is a semistructured interview that covers a range of events in the person's life and, for each stressor, covers the timing and broader life context. A number of major community and patient studies have documented that this in-depth focus provides a more predictive, reliable, and accurate assessment of threat than other available measures (Brown & Harris, 1989). In conducting interviews and ratings, attention is paid to three potential confounds in the measurement of life events: poor recall of event timing, biased evaluations of threat, and increased stress secondary to symptomatic behavior. Research suggests that individuals accurately remember severe events for at least 1 year (Brown & Harris, 1982). Raters are required to evaluate the context of each event in determining the severity. For example, for some women, pregnancy is a very desirable, planned goal. However, for a single teenager living in poverty, pregnancy can convey a much greater degree of threat.

On the basis of the event and the objective context, the raters evaluate the long-term threat for each event, or the severity for the average person 7-10 days after its initial occurrence. Threat ratings are made on a scale of 1 (*marked threat*) to 4 (*little or no threat*). Although rating threat requires weighing contextual information carefully, ratings of *marked threat* generally refer to events as severe as the loss of an immediate family member or a confidant, *moderate threat* ratings refer to events as severe as the loss of close friends or threat to immediate family relationships, and *some threat* ratings can include serious arguments and significant changes in core relationships. Within the LEDS, severe events have been empirically defined as those rated moderate or marked in threat. In addition, all events are rated for independence, or the extent to which an event was influenced by personality or psychopathology. For example, early signs of mania, such as irritability, are likely to increase marital strife. Raters are provided with details of how each event unfolded. Where there is ambiguity, they are asked to assume symptomatology and then to consider whether there is a possibility that the event could be due to symptomatology. Only independent events are used in primary analyses. The author has received training in the LEDS interview. All ratings of threat and independence were anchored using Brown's dictionaries, which provide tens of thousands of examples of ratings (Brown & Harris, 1978). Previous research has documented high inter-rater reliability for ratings of threat and independence.

The LEDS interviews investigated the presence of life events over the past year, prior to the start of treatment and at follow up. In the study life events with a moderate amount of threat or unpleasantness or a rating of 2 according to the Brown & Harris Scale were included. The total number of such life events was used as a continuous variable in the analysis.

5.7.2 Relapse

Relapse and recurrence of symptoms was estimated retrospectively using the SCID interview criteria for a single episode at the follow up assessments. In addition PANSS scoring criteria were applied to estimate symptom severity during these episodes.

A further measure of relapse and recurrence was the service related data that were collected for each participant. Recorded are any hospital admissions and emergency contacts due to deterioration in bipolar symptomatology. The number and length in days of any psychiatric inpatient admissions were collected as well as the number of psychiatric emergency appointments and routine psychiatric and CPN follow up appointments during the follow up period.

5.8 Statistical Analysis

All statistical analyses in this study were carried out with the software packages SPSS 15.0 for windows; and EQS 6.1 for covariance modelling in Chapter 8 only.

Treatment effects on key variables over time were analysed using repeated measures analysis of variance (ANOVA) and multivariate repeated measures analysis of variance (MANOVA). Post hoc comparisons between the different time points were calculated using Scheffe test, a Bonferroni correction was applied in accordance with the number of paired comparisons carried out. Categorical indicators relating to relapse and recurrence as well as variables referring to service use were compared using Chi² test statistics.

All analyses in Chapters 6 and 7 were carried out both on the sample of treatment completers N=174 participants and on an intent to treat sample including all participants starting treatment N= 196. On some measures there was a small proportion of missing data. Systematic comparisons of all DV and IV were carried out between completers and non-completers at all available time points. As there were no systematic significant differences in any of the variables missing data are assumed to be missing at random. Therefore, in the intent to treat analysis both regression models (R) and Expectation Maximisation (EM) methodology are employed to estimate missing data (Tabachnik & Fidell, 2007). As the two sets of results did not vary significantly the results of the Regression imputation are reported in the following sections as this method is understood to avoid bias introduced by the EM method not adding error in the imputed data set (Graham et al., 2003).

VI Treatment Outcomes

6.1 Sample characteristics at baseline

6.1.1 Demographic Characteristics

Table 6.1 presents the key demographic characteristics of the sample, comparing the two experimental groups. The sample here comprises all individuals who completed the baseline assessments.

Table 6.1: Baseline characteristics of the sample by treatment group

	Treatment Group (n=106)	Control Group (n=96)	
Age, mean (SD)	34.86 (9.65)	38.45 (11.30)*	$t(200)=-2.43$; $p=.016$
Gender, N (%)			$\chi^2(1)=0.46$; $p=.494$
Male	49 (46.2)	49 (51.0)	
Female	57 (53.8)	47 (49.0)	
Occupational status			$\chi^2(3)=4.80$; $p=.440$
Unemployed	54 (50.9)	40 (51)	
Employed	39 (36.8)	31 (32.3)	
In education	10 (9.4)	12 (12.5)	
Voluntary work	3 (2.8)	4 (4.2)	
Marital status			$\chi^2(2)=4.34$; $p=.362$
Married/ cohabiting	24 (22.6)	23 (24)	
Single	64 (60.4)	54 (56.2)	
Separated/ divorced	18 (17)	19 (19.8)	
Education			$\chi^2(2)=5.79$; $p=.327$
Higher education	63 (59.4)	51 (53.1)	
Job training	16 (15.1)	17 (17.8)	
No further education	27 (25.5)	28 (29.2)	

The only significant difference between the treatment plus TAU and the waiting list TAU group was that the average age was significantly lower in the treatment group ($t(200) = -2.43$; $p = .016$).

These baseline demographic variables have further been systematically compared between gender groups and groups allocated to the two treatment conditions, individual and group treatment. No significant differences between male and female participants were found apart from differences in marital status in that more women were married and/or cohabiting with their partners than men ($\chi^2(3) = 9.98$; $p = .041$). No significant differences were found between participants in the two treatment modalities.

6.1.2 Demographic Psychosocial Characteristics

Table 6.2 illustrates that there are no significant differences between the treatment plus TAU and TAU groups in terms of key social characteristics and early significant life stressors. Comparisons of these variables between female and male participants as well as between the two treatment modalities also yielded no significant differences in these variables; with the exception that more women in the trial were living with their children than men ($\chi^2(3) = 13.68$; $p = .028$).

Table 6.2: Baseline Social Network Characteristics by treatment group

	Treatment Group (n=106)	Control Group (n=96)	
Living with children N (%)	14 (13.2)	16 (16.6)	$\chi^2(3)=3.35$; $p= .500$
No of people in household	2.39 (1.55)	2.12 (1.14)	$t(163)=1.18$; $p= .237$
No of relatives	2.53 (2.75)	2.91 (3.45)	$t(162)=-.77$; $p= .442$
No of friends	8.39 (17.7)	7.41 (6.7)	$t(141)= .46$; $p= .671$
Childhood losses	84 (78.4)	81 (84.3)	$\chi^2(3)=2.87$; $p= .411$
Bereavement	21 (19.8)	23 (23.9)	
Family fragmentation	10 (9.4)	7 (7.3)	
Abuse	9 (8.5)	7 (7.3)	
Other	43 (40.5)	44 (45.8)	

6.1.3 Clinical Characteristics

Tables 6.3 and 6.4 summarise the baseline treatment and clinical characteristics for the treatment and TAU groups. Service related data were available for 188 (97%) of the sample.

Of the whole sample 95.1 % received a diagnosis of Bipolar I Disorder, 2.9% or 6 individuals received a diagnosis of Bipolar II disorder, of those 4 did not commence treatment. 16% of the sample, or 33 individuals had a co-morbid anxiety disorder and 14% or 29 individuals had a co-morbid psychotic disorder. 64% of participants reported in the assessment interview that they engaged in regular or occasional recreational drug use, mainly cannabis, but also amphetamines and cocaine. No participants were included who abused opiates. Within the scope of the study it proved impossible to gain a reliable measure of substance use and no systematic comparisons including substance use variables were included.

The only significant difference that emerged between the treatment and TAU groups is that participants in the control group spend less days in hospital following an inpatient admission than the participants who were allocated to the treatment group ($t(187)=2.27$; $p=.024$).

There are no significant differences in the history of illness and service use related variables between men and women at baseline. Also, treatment modality does not differentiate in respect of these variables.

Table 6.3: History of illness and service use by treatment group

	Treatment Group (n=106)	Control Group (n=96)	
Age 1 st mental health problems, mean (SD)	21.65 (8.58)	22.91 (9.04)	$t(197)=-.752$; $p=.453$
Age BD diagnosis	28.97 (9.11)	31.12 (10.50)	$t(200)=-.752$; $p=.453$
No of depressed episodes (total)	11.45 (19.42)	14.34 (28.31)	$t(197)=-.75$; $p=.453$
No of (Hypo)manic episodes (total)	10.21 (13.81)	6.60 (10.38)	$t(197)=1.78$; $p=.076$
No of hospital admissions 18 M prior			$\chi^2(3)=13.25$; $p=.103$
0	45 (42.4)	52 (54.1)	
1	22 (20.7)	29 (30.2)	
2	19 (17.9)	16 (16.6)	
>2	10 (9.4)	0 (0)	
Days spend in hospital 18 M prior to treatment	25.72 (35.37)	15.56 (22.37)*	$t(187)=2.27$; $p=.024$
Mental Health contacts in past 12 months			
CPN	14.60 (11.65)	12.74 (12.15)	$t(187)=0.95$; $p=.340$
Psychiatrist	8.76 (6.04)	7.64 (5.29)	$t(187)=0.96$; $p=.337$
Others	1.21 (1.74)	2.92 (7.75)	$t(187)=-.894$; $p=.373$

Differences in key clinical indicators from the observer rated and self report clinical measures between the two groups are outlined in Table 6.4. A preliminary examination of these variables showed that some are not normally distributed (the positive symptom scores on PANSS and BPRS) and the significance tests are reported for the transformed and normally distributed variables. A closer examination of the distributions and other variable characteristics will be illustrated as part of the main analysis below.

Table 6.4: Clinical characteristics by treatment group

	Treatment Group (n=106)	Control Group (n=96)	
Positive and Negative Symptom Scale (PANSS)			
General	28.6 (6.6)	27.5 (6.3)	t(200)=1.25; p= .210
Positive	9.1 (3.5)	8.5 (2.3)	t(200)=1.50; p= .134
Negative	13.7 (5.2)	12.6 (5.3)	t(200)=1.61; p= .108
Brief Psychiatric Rating Scale (BPRS)			
General	37.4 (6.5)	37.2 (8.5)	t(200)=0.202; p= .840
Depression/ anxiety	61.4 (12.4)	62.4 (22.1)	t(200)=0.708; p= .480
Beck Depression Inventory (BDI-II)	21.2 (13.2)	20.4 (14.5)	t(200)=1.001; p= .318
Altman Mania Scale (AMS)	2.8 (3.4)	2.9 (3.7)	t(184)=0.722; p= .472
Bech Rafealsen Mania Scale (BRMS)	3.6 (3.0)	3.1 (3.3)	t(184)=0.933; p= .352
Bipolar Longitudinal Investigation of Symptoms (BLIP)			
Mania	9.9 (8.3)	8.9 (9.5)	t(184)=0.196; p= .845
Depression	14.6 (13.4)	13.9 (14.4)	t(184)=0.584; p= .560
Mixed Symptoms	22.16 (17.8)	22.28 (16.0)	t(184)= 0.526; p= .959
Quality of Life (WHOQOL-BREF)			
Domain 1	11.3 (2.8)	11.7 (2.9)	t(200)=1.03; p= .302
Domain 2	11.3 (2.6)	11.8 (2.7)	t(200)=1.29; p= .197
Domain 3	12.0 (3.0)	13.1 (2.7)*	t(200)=2.48; p= .014
Domain 4	13.5 (2.5)	14.5 (2.1)**	t(200)=3.25; p= .001

As illustrated in Table 6.4, the only significant differences between the two groups are in the Quality of Life measure (WHOQOL-BREF), specifically in Domain 3, social relationships, and Domain 4, environment, where participants in the control group have significantly higher scores at baseline compared with the treatment group. On all measures of symptom severity and distress there are no significant differences between the two groups at baseline.

6.2 Statistical Analysis

The main treatment effect between the treatment condition (CIT+TAU) and the waiting list control treatment as usual condition (TAU) were analysed using two way factorial multivariate analysis of variance (MANOVA) and two way factorial analysis of variance (ANOVA). The effect sizes of the main effects will be reported as partial eta squared (η_p^2).

Treatment effect in terms of symptom severity over the five time points were analysed using repeated measures analysis of variance (ANOVA) and multivariate repeated measures analysis of variance (MANOVA). Post hoc comparisons between the different time points were calculated using Scheffe test, a Bonferroni correction was applied in accordance with the number of paired comparisons carried out. Categorical indicators relating to relapse and recurrence as well as variables referring to service use were compared using Chi² test statistics.

6.2.1 Assumptions

Prior to the calculations of the primary outcomes of the trial key variables have been examined regarding their normal distribution and homogeneity of variance. In the repeated measures analyses the assumption of sphericity was further investigated.

Variables relating to positive symptoms (BPRS and PANSS subscales of positive symptoms) showed moderate indications of positive skewness (z-value for skewness above 1.96). These variables have been transformed using their logarithm prior to the analysis.

6.3 Trial outcome – Intent to Treat Analysis

Is psychological treatment and treatment as usual more effective in improving quality of life, reducing psychiatric symptoms and reducing indicators of relapse than treatment as usual alone?

The principal hypothesis addresses the question whether psychological intervention and TAU does have a positive effect on overall quality of life, symptom severity, and indicators of relapse when compared to TAU alone.

The secondary hypothesis is whether any treatment effects are due to a specific treatment modality chosen by the participants.

The effectiveness of the psychological intervention plus TAU versus treatment as usual alone was analysed using two way factorial repeated measures ANOVA and two way factorial repeated measures MANOVA. The primary outcome was in relation to quality of life (WHOQOL-BREF). Secondary outcomes related to psychiatric symptomatology and emotional distress as measured by PANSS and BPRS and the clinical self-report measures (BDI, BLIP, and Mania Scales). Effect sizes are reported as partial eta squared (η_p^2) (Tabachnik & Fidell, 2007) and interpreted using standard convention for the interpretation of effect size (Olejnik & Algina, 2000; Cohen, 1988,1992).

To aid the clarity of presentation the hypothesis will be examined in sections, differentiating primary and secondary outcomes and separating the observer rated measures, self report measures of symptom severity, and service data.

6.3.1 Main treatment effect for primary outcome – Quality of Life

Changes in perceived quality of life were subjected to a two way repeated measures multivariate analysis of variance (MANOVA) with two time points, at baseline and after treatment or end of waiting period. This was carried out in two groups, psychological treatment and treatment as usual (CIT+TAU) and treatment as usual alone (TAU).

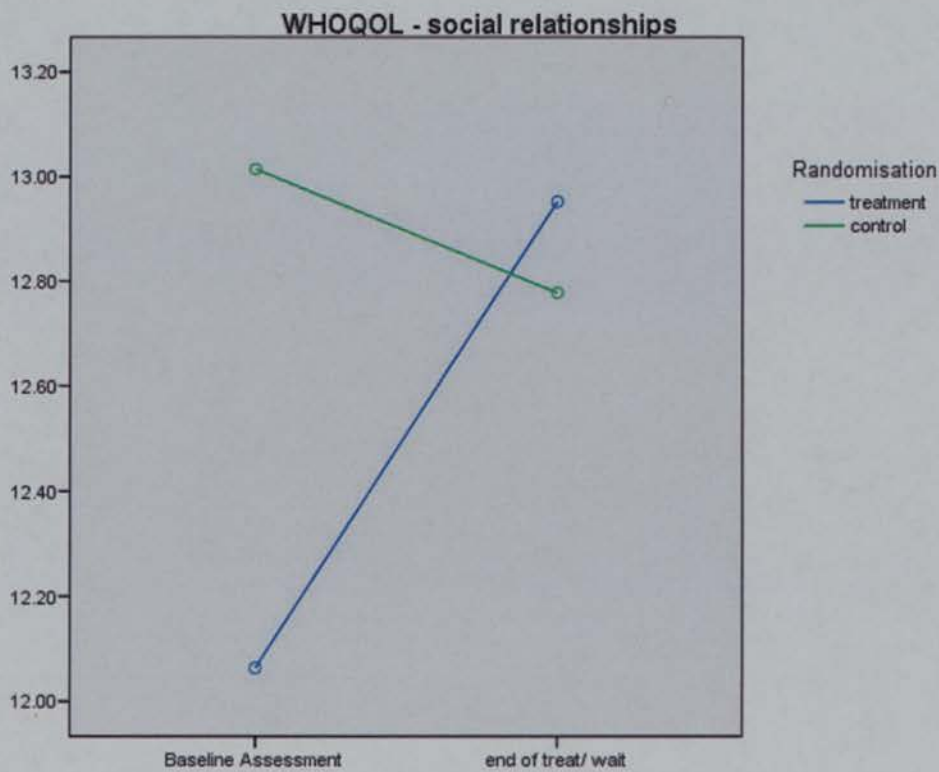
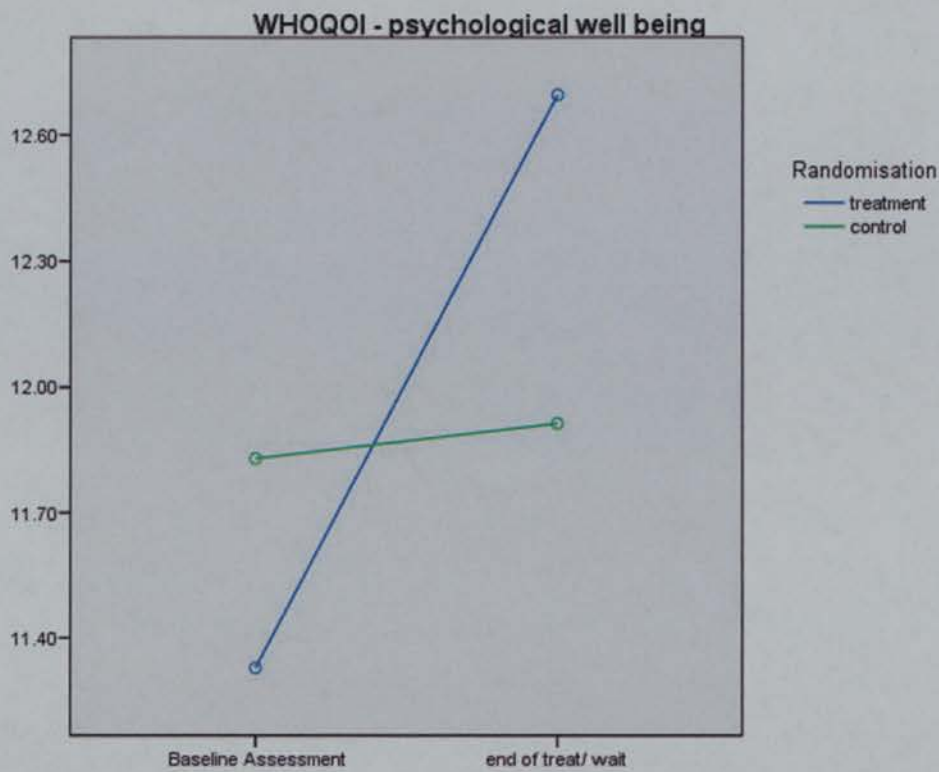
With the use of Wilks' criterion, the combined dependent variables of Quality of Life (WHOQOL-BREF Dimensions) were significantly affected over the two time points, $F(3,370) = 4.92$, $p = .002$, but not by group, $F(3,370) = 1.59$, $p = .191$; there were significant interaction effects of time by treatment, $F(3,370) = 3.78$, $p = .011$. The effect size of the interaction effect is $\eta_p^2 = .32$, which is equivalent to a large effect size.

In order to test the impact of each main effect on the individual quality of life domain, univariate analysis of variance was performed on the individual dependent variables. As illustrated in Table 6.5 this effect applies to three quality of life domains, psychological well being, social relationships and environment, but not to physical health. This indicates that the change over time in quality of life showed a significant advantage for CIT+TAU when compared with TAU alone over the same time period.

Table 6.5: Psychological Treatment & TAU versus TAU – Quality of Life

	CIT + TAU		TAU		F Time*group interaction
	Before M (SD)	After M (SD)	Before M (SD)	After M (SD)	
Quality of Life (WHOQOL-BREF)					
Domain 1: Physical health	11.33 (2.87)	12.79 (3.09)	11.79 (2.93)	12.23 (3.18)	n.s.
Domain 2: Psychological well-being	11.32 (2.67)	12.69 (2.78)	11.82 (2.73)	11.91 (2.91)	F(1,372)=5.02, p= .020
Domain3: Social Relationships	12.06 (3.06)	12.95 (3.17)	13.01 (2.72)	12.77 (2.79)	F(1,372)=3.39, p= .050
Domain 4: Environment	13.48 (2.50)	15.16 (2.22)	14.52 (2.11)	14.60 (2.35)	F(1,372)=11.22, p< .001

Figure 6.1 illustrates the interaction effect in the prioritised quality of life domains. In all three WHOQOL-BREF domains participants in the treatment group (CIT+TAU) started off with significantly lower quality of life scores when compared with the control group (TAU). After the intervention, participants in the treatment group showed a significant increase in the quality of life scores above the mean scores of the control group, consequently demonstrating a positive interaction effect of the treatment condition on quality of life over that time period.



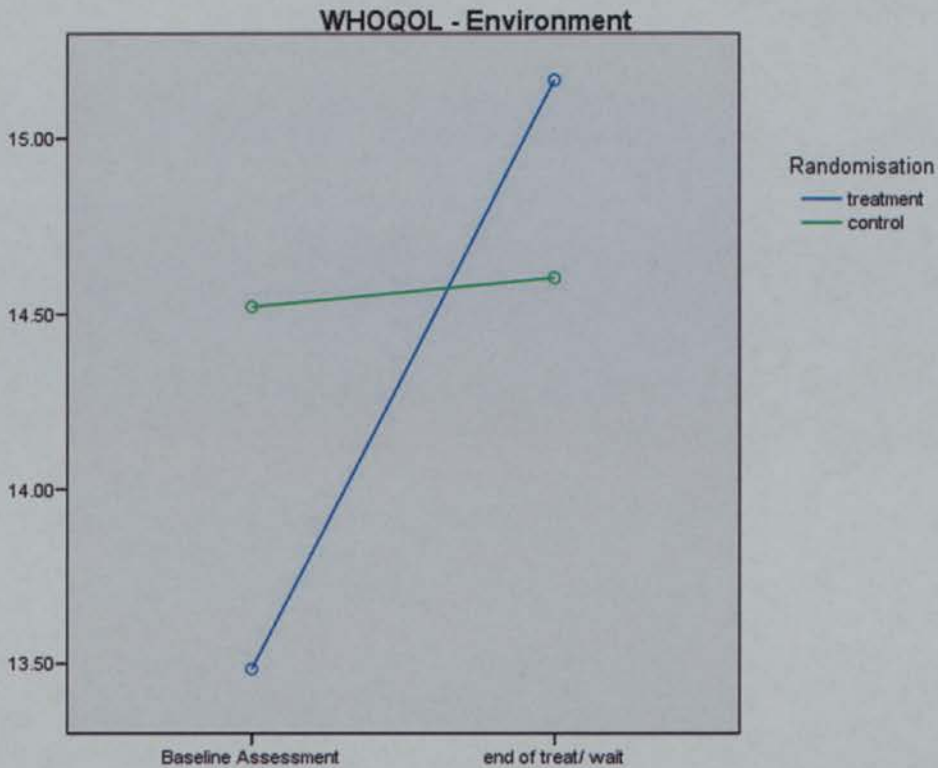


Figure 6.1: Time x Group – Quality of Life

6.3.2 Main treatment effects for secondary outcomes – Observer rated measures of symptom severity

Did psychological treatment plus treatment as usual have a beneficial effect on psychiatric symptoms over and above the effects of treatment as usual alone? In order to test the secondary outcomes relating to observer rated and self report measures of psychiatric and bipolar symptom severity, a number of two way repeated measures multivariate analyses of variance and two way repeated measures analyses of variance were performed.

For the Brief Psychiatric Rating Scale (BPRS) there was a significant time effect, $F(5,374) = 15.86$, $p < .001$, and a significant group effect, $F(5,374) = 8.17$, $p < .001$. There were also

significant time by group interaction effects for negative symptoms, positive symptoms and the BPRS depression dimension, $F(5,374) = 5.75$, $p < .001$. The effect size of the interaction effect is $\eta_p^2 = .09$, which is equivalent to a lower band medium effect size.

Likewise, for the Positive and Negative Symptoms Scale (PANSS) there was a significant time effect, $F(5,376) = 26.43$, $p < .001$, and a significant group effect, $F(5,376) = 6.96$, $p < .001$. There were also significant time by group interaction effects for negative symptoms, positive symptoms and the BPRS depression dimension, $F(5,376) = 15.15$, $p < .001$. The effect size of the interaction effect is $\eta_p^2 = .18$, which is equivalent to a medium effect size.

Table 6.6 highlights the significant symptom dimensions of the interaction effect in their univariate analyses of variance computations. All key BPRS dimensions, general psychiatric symptoms, depression, negative symptoms and positive symptoms show significant time by treatment group interaction effects. Equally all three PANSS dimensions, general symptoms, positive symptoms and negative symptoms show a significant interaction effect.

Table 6.6: Psychological Treatment & TAU versus TAU – observer rated measures of psychiatric symptoms

		CIT + TAU		TAU		F Time*group interaction
		Before M (SD)	After M (SD)	Before M (SD)	After M (SD)	
BPRS						
	general	37.21 (6.53)	28.54 (4.40)	37.20 (8.53)	35.49 (6.82)	F(1,378)=25.30, P< .001
	depression	61.40 (12.40)	44.15 (9.52)	62.46 (22.08)	58.50 (14.41)	F(1,378)=17.89, P< .001
	positive symptoms	8.70 (2.33)	7.68 (1.38)	8.46 (2.18)	8.32 (2.65)	F(1,378)=3.89, p= .045
	negative symptoms	5.83 (1.61)	4.34 (0.70)	5.67 (1.64)	5.21 (1.37)	F(1,378)=12.63, p< .001
PANSS						
	general	28.63 (6.63)	20.06 (3.42)	27.52 (6.34)	26.50 (6.00)	F(1,378)=42.18 P< .001
	positive symptoms	9.18 (3.51)	7.47 (1.26)	8.45 (2.29)	8.34 (2.32)	F(1,378)=9.48, p= .002
	negative symptoms	13.74 (5.22)	7.94 (1.80)	12.68 (5.28)	11.73 (4.56)	F(1,378)=27.83, P< .001

Figure 6.2 illustrates the clear treatment time and group interaction effects for the severity of psychiatric symptoms as measured by the BPRS and PANSS observer rated scales. For the general, depressive and negative symptom dimension there appears to be a clear positive effect on symptom reduction for the CIT+TAU group. For the two measures of positive symptoms there seems to be a cross over effect in that participants in the CIT+TAU treatment groups show a relatively higher symptom level at baseline when compared with the control group and a clear symptom reduction at the end of treatment. This suggests that the psychological intervention had a clear impact on manic and psychotic symptoms in this group.

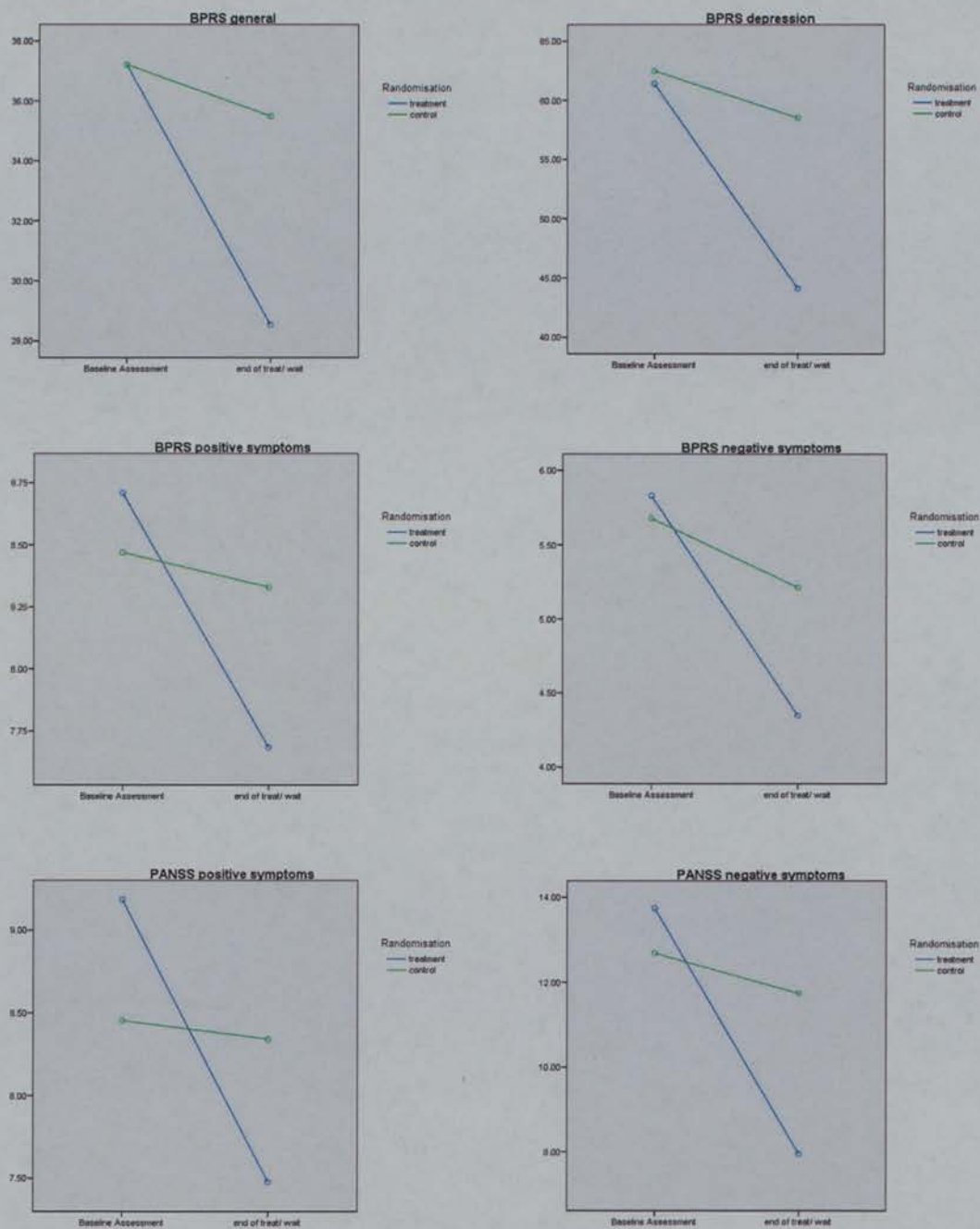


Figure 6.2: Time x Group – observer rated psychiatric symptoms

6.3.3 Main treatment effects for secondary outcomes – Self-report measures of symptom severity

In the following section the effects of psychological intervention plus TAU on self report measures of psychiatric and bipolar symptoms and emotional distress are reported when compared to TAU alone.

On the Beck Depression Inventory (BDI-II) scores the two way analysis of variance yielded a main effect for time, $F(1,379) = 29.08$, $p < .001$, such that the average score was significantly lower at the second timepoint. The main effect of group was not significant, $F(1,379) = 2.51$, $p = .113$. However, there was a significant interaction effect, $F(1,379) = 9.25$, $p = .003$, indicating that the treatment group had a significantly greater reduction in their BDI-II depression scores at timepoint two when compared to the control group. This effect is illustrated in Figure 6.3. The effect size of $\eta_p^2 = .25$ indicates a large effect size for this variable.

For the two mania scales it can be reported that in the two way repeated measures ANOVA's there are no significant effects for the main effects or the interaction effect for the Altman Mania Scale. For the Bech Rafealsen mania scale there is a significant main effect of time, $F(1,379) = 4.51$, $p = .033$, but not for group, $F(1,379) = 1.71$, $p = .164$, or time group interaction, $F(1,379) = 3.74$, $p = .054$, in that there is a significant reduction in mania symptoms as assessed by this measure between baseline and timepoint two. Similarly for the BLIP scale the two way repeated measure MANOVA shows a significant effect over the two timepoints, $F(2,379) = 8.75$, $p < .001$, but no main group effect, $F(2,379) = 0.26$, $p = .770$, or time by group interaction effect, $F(2,379) = 1.55$, $p = .212$.

These time effects on the BLIP measure are illustrated in Figure 6.4. Table 6.7 highlights the univariate differences for the interaction of treatment groups and time.

Table 6.7: Psychological Treatment & TAU versus TAU – observer rated measures of psychiatric symptoms

		CIT + TAU		TAU		F Time*group interaction
		Before M (SD)	After M (SD)	Before M (SD)	After M (SD)	
BDI-II		21.97 (13.23)	10.42 (11.61)	20.40 (14.57)	17.02 (14.59)	$F(1,380) = 8.89$, $p = .003$
BLIP						
	depression	14.68 (13.40)	8.20 (10.22)	13.93 (14.48)	10.77 (11.56)	n.s.
	mania	9.97 (8.36)	6.34 (7.37)	8.95 (9.56)	7.90 (8.90)	n.s.
	mixed symptoms	24.66 (16.36)	14.54 (13.28)	22.89 (19.65)	18.67 (17.07)	n.s.
Bech Rafealsen mania scale		3.65 (3.00)	2.78 (3.21)	3.16 (3.38)	2.80 (2.62)	$F(1,380) = 4.74$, $p = .049$

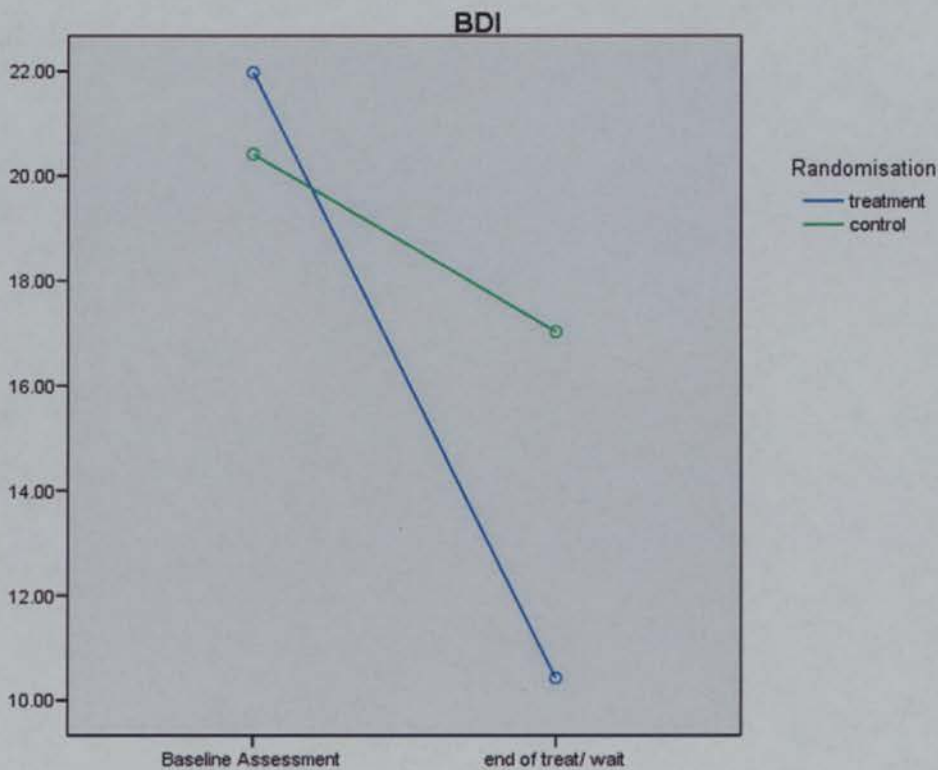


Figure 6.3: Time x Group – BDI-II

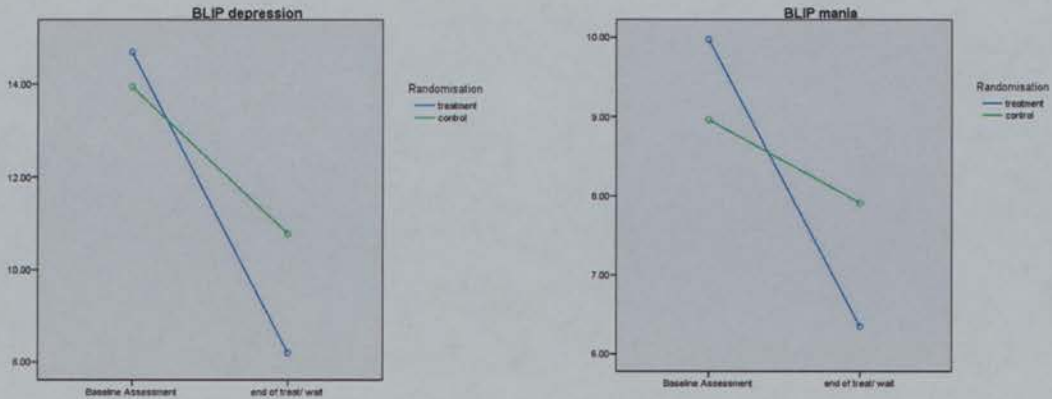


Figure 6.4: Time x Group – BLIP mania and BLIP depression

6.3.4 Gender effects on main treatment effect

In order to test whether there are systematic differences in the main treatment effects between men and women in the treatment trial, gender was added as a covariate in the two way repeated measures ANOVA and two way repeated measures MANOVA calculations, and the interaction effect of time by gender was analysed.

For the primary outcome variables, the four dimensions of quality of life (WHOQOL-BREF), a two way multivariate analysis of variance yielded no significant main effect for gender, $F(4,365) = 1.48$, $p = .206$. There was also no significant interaction effect of time by gender $F(4,365) = 1.67$, $p = .607$. The significant interaction effect of time by treatment group reported above persisted $F(4,365) = 3.52$, $p = .008$.

In terms of the observer rated measures of severity of psychiatric symptoms (BPRS & PANSS), there were similar findings. On the PANSS, a two way repeated measure MANOVA showed a significant main effect for gender $F(3,372) = 7.46$, $p < .001$, but no

significant interaction effect of time by gender $F(3,372) = 1.14$, $p = .333$. The interaction effect of time by treatment remained highly significant $F(3,372) = 10.02$, $p < .001$. In parallel, on the BPRS, a two way repeated measure MANOVA also showed a significant main effect for gender $F(3,372) = 8.26$, $p < .001$, but no significant interaction effect of time by gender $F(3,372) = 0.77$, $p = .510$. The interaction effect of time by treatment remained significant $F(3,372) = 14.11$, $p < .001$.

For the self report measures of depression (BDI-II) and bipolar symptoms (BLIP), there were no effects of gender on the treatment effect. For the BDI-II a two way repeated measure analysis of variance yielded no significant main effect of gender, $F(1,376) = 2.20$, $p = .139$. There was also no significant interaction effect of time by gender $F(1,376) = 0.06$, $p = .807$. The significant interaction effect of time by treatment group persisted with the inclusion of gender as a covariate $F(1,376) = 5.91$, $p = .003$. On the BLIP, a two way repeated measure multivariate analysis of variance showed a significant main effect of gender, $F(2,375) = 3.23$, $p = .040$. But there was no significant interaction effect of time by gender $F(2,375) = 1.05$, $p = .346$. As reported above there was no significant interaction effect of time by treatment group for this measure $F(2,375) = 1.94$, $p = .145$.

The results on the gender effects as a covariate of the main treatment effects therefore show that there are some group differences between men and women in both the treatment and control groups. These are mainly in relation to severity scores of depression, both in observer rated and self-report measures at baseline, in that women show higher depression scores than men, and in relation to the severity of psychotic symptoms, that at baseline men demonstrate a higher level of severity on these dimensions. These gender group differences do not influence the main treatment effects between the treatment and control group.

6.4 Outcome Indicators Following Psychological Intervention

Apart from the main treatment effects in comparing psychological intervention plus TAU with TAU alone, an essential question for the effectiveness of psychological intervention for bipolar disorder is whether the key treatment effects were maintained over time. Did the course of integrated psychological therapy reduce measures of symptom severity and distress following treatment and at follow up?

Further, did patient choice in treatment modality impact on the clinical outcomes? Or, was any treatment effect dependent upon the treatment modality chosen by the trial participants?

6.4.1 Outcomes – Quality of Life

The results for the repeated measures ANOVA for the primary outcome variable of Quality of Life (WHOQOL-BREF) are detailed in Table 6.8. The quality of life domains yielded medium effect sizes for changes over time, between $\eta_p^2 = .12$ and $\eta_p^2 = .16$ (Olejnik & Algina, 2000).

Table 6.8: Repeated Measure ANOVA's for quality of life (WHOQOL-BREF)

Variable within subject effects	F (df); sig Time	F (df); sig Time*TreatmentMode
Quality of Life (WHOQOL-BREF)		
Domain 1	10.01** (3,189); $p < .001$ *	0.33 (3,189); $p = .804$ *
Domain 2	35.33** (3,189); $p = .001$	1.26 (3,189); $p = .285$
Domain 3	8.08** (3,189); $p < .001$	1.97 (3,189); $p = .116$
Domain 4	10.80** (3,189); $p < .001$ *	0.99 (3,189); $p = .399$ *

*Sphericity not assumed (Greenhouse-Geisser correction applied)

As outlined above the perceived quality of life improved following intervention and that effect could be maintained at follow up. The within subject comparisons show that there are slightly different patterns of change for the four domains of the WHOQOL-BREF.

Table 6.9: Pairwise comparisons of WHOQOL-BREF domain 1 over time

Time		Dom 1 Mean difference	Std. Error	Sig
1	2	-.1253**	.307	.000
	3	-.631*	.307	.041
	4	-.1485**	.256	.000
2	3	.622	.332	.063
	4	-.232	.281	.410
3	4	-.854**	.301	.005

Time 1: start of treatment	Time 2: end of treatment
Time 3: follow up 6 months	Time 4: follow up 18 months

Table 6.10: Pairwise comparisons of WHOQOL-BREF domain 2 over time

Time		Dom 2 Mean difference	Std. Error	Sig
1	2	-.992**	.238	.000
	3	-.632*	.257	.015
	4	-.788*	.270	.004
2	3	.360	.273	.188
	4	.204	.246	.407
3	4	-.156	.268	.560

Time 1: start of treatment	Time 3: follow up 6 months
Time 2: end of treatment	Time 4: follow up 18 months

Table 6.11: Pairwise comparisons of WHOQOL-BREF domain 3 over time

Time		Dom 3 Mean difference	Std. Error	Sig
1	2	-.516	.266	.054
	3	-.174	.301	.563
	4	-1.271**	.272	.000
2	3	.342	.305	.264
	4	-.755*	.263	.005
3	4	-1.097**	.271	.000

Time 1: start of treatment	Time 3: follow up 6 months
Time 2: end of treatment	Time 4: follow up 18 months

Table 6.12: Pairwise comparisons of WHOQOL-BREF domain 4 over time

Time		Dom 4 Mean difference	Std. Error	Sig
1	2	-1.039**	.199	.000
	3	-1.103**	.255	.000
	4	-1.189**	.247	.000
2	3	-.064	.235	.785
	4	-.150	.229	.511
3	4	-.086	.271	.751

Time 1: start of treatment	Time 3: follow up 6 months
Time 2: end of treatment	Time 4: follow up 18 months

For Domain 1, physical well being, there is a clear improvement in perceived quality of life between the start and end of treatment, which was maintained at 18 months follow up. Domain 2, psychological well being, yielded a similar result. For Domain 3, social relationships, the treatment effects are different, in that there is no significant improvement in this dimension between the beginning of treatment to follow up at six months, but a significant positive change between follow up at six months and follow up at 18 months. Domain 4, environmental domain, shows a steady and continued

improvement over all time points. Details of these comparisons are detailed in Tables 6.9 to 6.12. Figures 6.5 and 6.6 illustrate patterns of change of Domain 2, psychological well being, and Domain 3, social relationships.

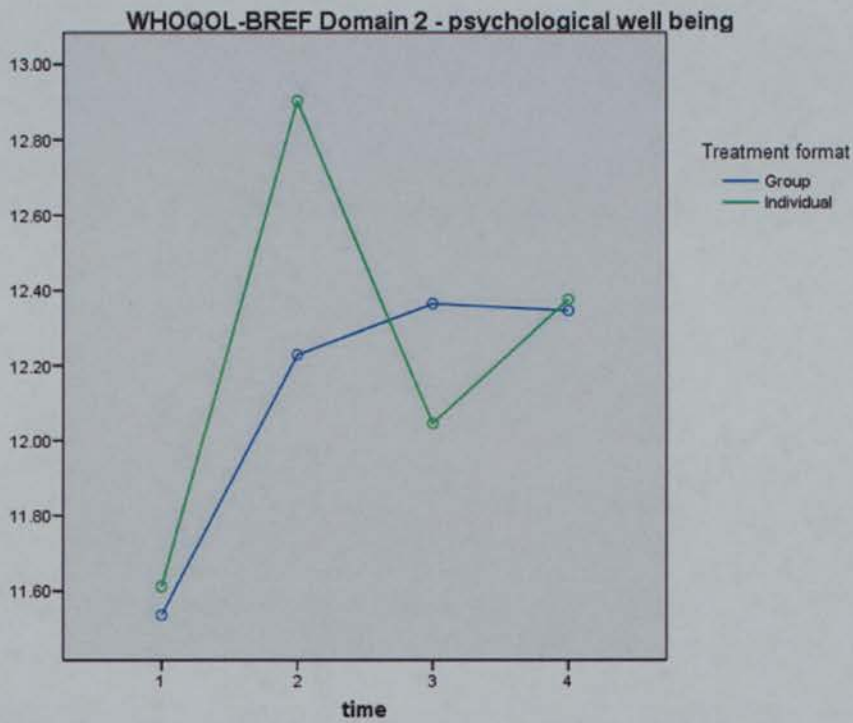


Figure 6.5: Quality of life scores over time – psychological well being

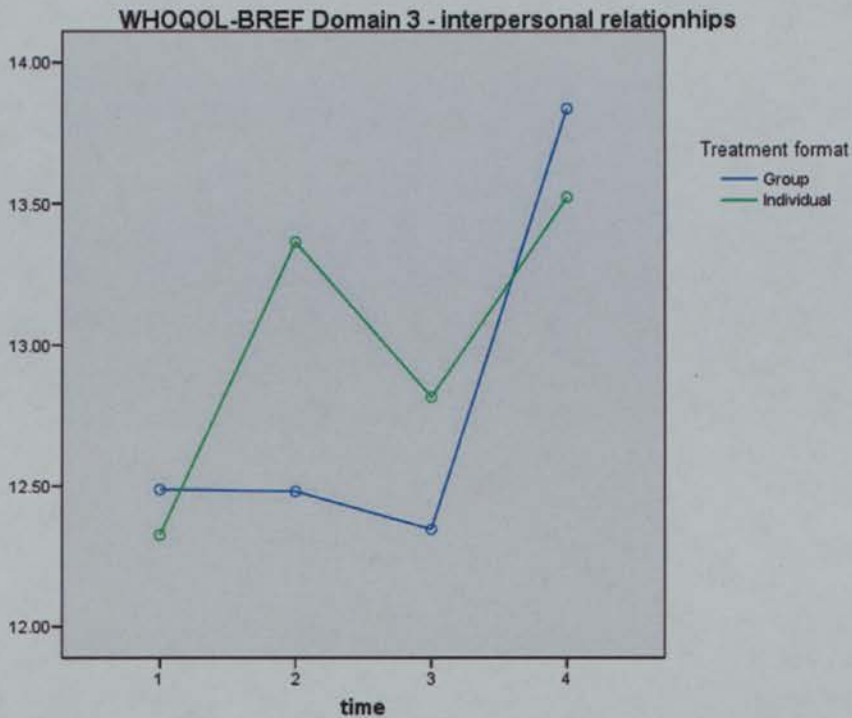


Figure 6.6: Quality of life scores over time – social relationships

6.4.2 Outcomes – Observer rated measures of symptom severity

The results for the repeated measures ANOVA for the relevant scores of the observer rated symptom measures over the four assessed time points are illustrated in Table 6.13. The general PANSS and BPRS scores are reported together with those subscale scores that have a particular mood and impairment rating. Where the condition of sphericity was violated according to the Mauchly's W test the Greenhouse-Geisser corrections were applied.

The effects for both general scores BPRS overall and PANSS general yielded large effect sizes of $r=0.59$ and $r=0.54$ respectively, whereas the sub scale scores of PANSS negative and BPRS depression/ anxiety yielded medium effect sizes of $r=0.38$ and $r=0.33$ (Cohen, 1992).

Table 6.13: Repeated measures ANOVA's for observer rated measures of symptom severity

Variable within subject effects	F (df); sig Time	F (df); sig Time*TreatmentMode
Positive and Negative Symptom Scale (PANSS)		
General	91.94** (3,189); p< .001	0.95 (3,189); p= .406
Negative	40.15** (3,189); p< .001	0.91 (3,189); p= .433
Brief Psychiatric Rating Scale (BPRS)		
General	96.35** (3,189); p< .001	0.49 (3,189); p= .721
Depression/ anxiety	80.42** (3,189); p< .001*	0.56 (3,189); p= .634*

*Sphericity not assumed (Greenhouse-Geisser correction applied)

As the repeated measures ANOVA's show there was a significant reduction in severity of symptoms as measured by the observer rated assessments following treatment. This change was further maintained at follow up at 6 months and 18 months after end of treatment. The tests of interaction with treatment modality were not significant, indicating that participants in either treatment modality experienced similar reduction in symptoms which was maintained at follow up. Table 6.14 and 6.15 outline the pairwise comparisons of the mean differences of the general BPRS and PANSS scores over time. These differences are further illustrated in Figures 6.7 and 6.8.

The only exception in this pattern is for the PANSS negative symptom sub scale where significant differences were found between end of treatment and both follow up points. Where there is a significant increase in symptoms following end of treatment. This indicates that the overall treatment effect for this subscale is maintained in that at follow up scores are significantly lower than a start of treatment, but that there is a significant relative increase in scores between end of treatment and follow up.

Table 6.14: Pairwise comparisons of PANSS general score over time points

Time		PANSS Mean difference	Std. Error	Sig
1	2	7.04**	.541	.000
	3	8.55**	.512	.000
	4	6.03**	.663	.000
2	3	1.50**	.412	.000
	4	-1.09	.604	.096
3	4	-2.51**	.557	.000

Time 1: start of treatment

Time 2: end of treatment

Time 3: follow up 6 months

Time 4: follow up 18 months

Table 6.15: Pairwise comparisons of BPRS general score over time points

Time		BPRS Mean difference	Std. Error	Sig
1	2	7.24**	.580	.000
	3	8.39**	.610	.000
	4	7.43**	.598	.000
2	3	1.15*	.534	.032
	4	.187	.500	.708
3	4	-.967	.520	.064

Time 1: start of treatment

Time 2: end of treatment

Time 3: follow up 6 months

Time 4: follow up 18 months

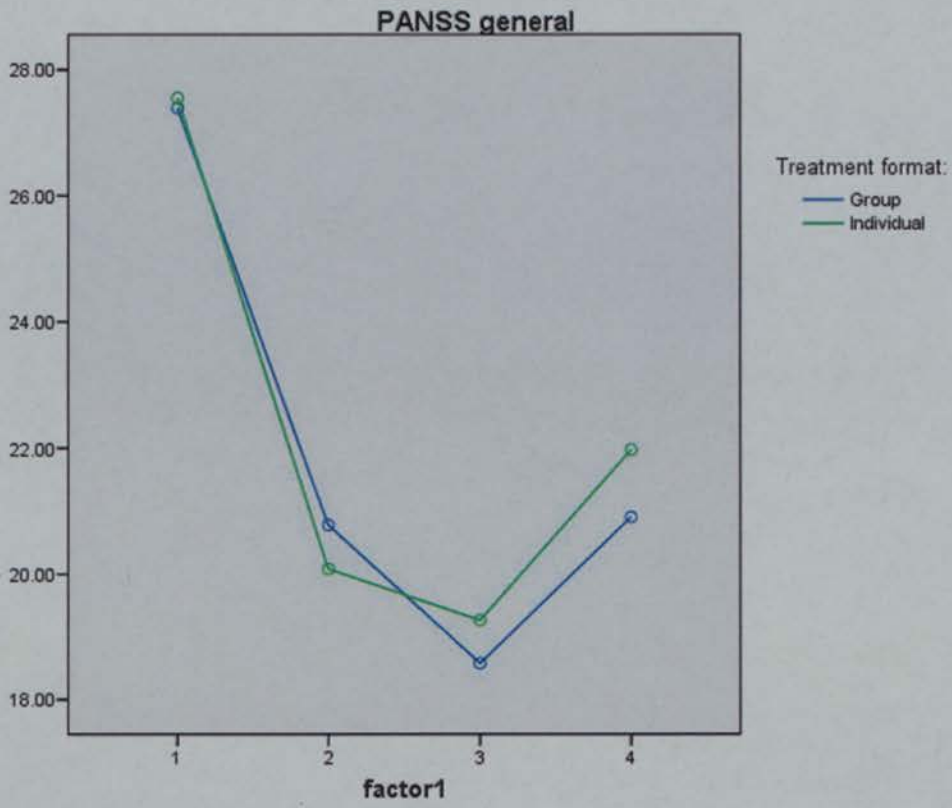


Figure 6.7: PANSS scores over time

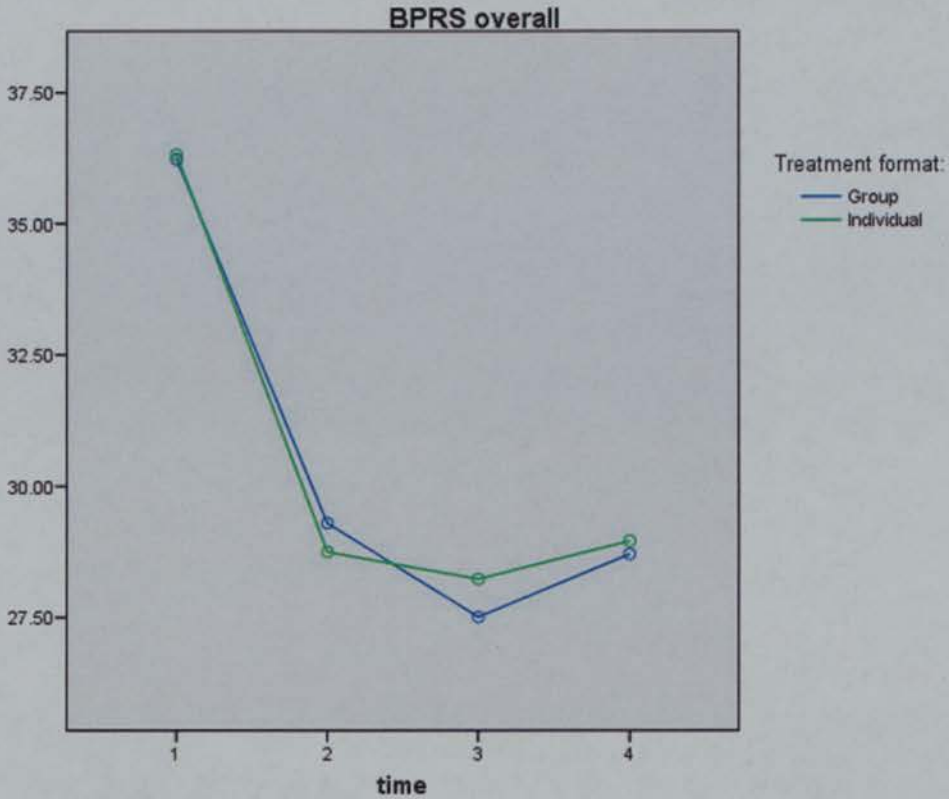


Figure 6.8: BPRS scores over time

6.4.3 Outcomes – Self report measures of symptom severity

In parallel to the analyses carried out for the observer rated measures of symptom severity similar analyses were conducted for the self-report scales of severity of symptoms, distress and quality of life. As above the main effect of repeated measures over time was investigated in interaction with treatment modality in order to explore whether any treatment effects in these areas are specific to the mode of treatment.

The overall results of the repeated measures ANOVA's for the self-report measures are reported in Table 6.16. The differences over time produced large to medium effect sizes for the self-report symptom measures: BDI, $r=.39$; AMS, $r=.23$; BLIP, D $r=.28$; BLIP, M

$r=.19$; BLIP Mixed $r=.27$. For the quality of life measure effect sizes were more modest, between $r=.12$ and $r=.16$.

Table 6.16: Repeated measures ANOVA's for self report measures of symptom severity and quality of life

Variable within subject effects	F (df); sig Time	F (df); sig Time*TreatmentMode
Beck Depression Inventory (BDI-II)	31.07** (4,188); $p < .001$ **	0.84 (4,188); $p = .497$ **
Altman Mania Scale (AMS)	2.06 (4,188); $p = .090$ **	1.72 (4,188); $p = .142$ **
Bech Rafealsen Mania Scale (BRMS)	4.96** (4,188); $p = .001$ **	1.08 (4,188); $p = .300$ **
Bipolar Longitudinal Investigation of Symptoms (BLIP)		
Mania	13.50** (4,188); $p < .001$	1.19 (4,188); $p = .276$
Depression	14.64** (4,188); $p < .001$ **	1.70 (4,188); $p = .147$ **
Mixed Symptoms	23.45** (4,188); $p < .001$	1.48 (4,188); $p = .204$

**Sphericity not assumed (Greenhouse-Geisser correction applied)

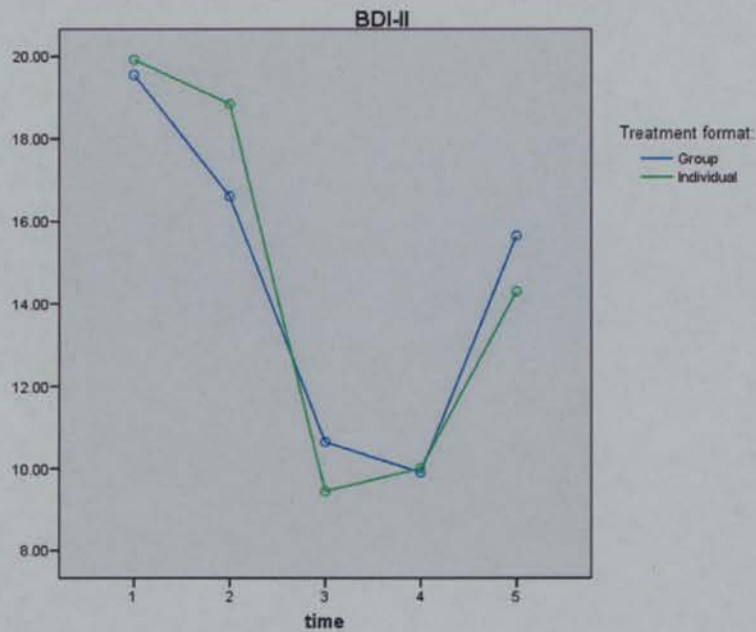
The within subject pairwise comparisons revealed that the depression score on the BDI-II (Table 6.17) and the mania scores on the AMS were significantly reduced between start of treatment and end of treatment, this effect was maintained at follow up at six months, but there was a significant increase in depression at follow up at 18 months from end of treatment. This final score however was still significantly lower than at start of treatment.

Table 6.17: Pairwise comparisons of BDI-II over time points

Time		BDI-II Mean difference	Std. Error	Sig
1	2	2.014	1.136	.078
	3	2.014**	1.130	.000
	4	9.779**	1.141	.000
	5	4.763**	1.261	.000
2	3	7.672**	1.058	.000
	4	7.765**	1.124	.000
	5	2.749*	1.246	.029
3	4	.093	.978	.924
	5	-4.923**	1.133	.000
4	5	-5.016**	.996	.000

Time 1: start of treatment
Time 3: end of treatment
Time 5: follow up 18 months

Time 2: mid treatment
Time 4: follow up 6 months

**Figure 6.9: BDI scores over time**

A similar pattern emerged for the Bech Rafealsen Mania Scale. There are significant improvements in scores between start and end of treatment that are maintained at the

six month follow up assessment but could not be maintained at 18 month follow up. There is a significant increase in mania scores between 6 and 18 months follow up and the scores at 18 months are no longer significantly lower than at start of treatment.

In terms of the Bipolar Longitudinal Investigation of Problems scale (BLIP) we can observe a steady decline in depression, mania and mixed symptom scores over time as detailed in Tables 6.18 and 6.20 and Figures 6.10 and 6.12 respectively. The overall treatment effect was maintained at 18 months follow up.

Table 6.18: Pairwise comparisons of BLIP mania over time points

Time		BLIP M Mean difference	Std. Error	Sig
1	2	1.103	.894	.219
	3	3.201**	.784	.000
	4	2.924**	.673	.000
	5	5.107**	.723	.000
2	3	2.098*	.905	.022
	4	1.820*	.894	.043
	5	4.004**	.819	.000
3	4	-.277	.649	.670
	5	1.906*	.616	.002
4	5	2.183**	.563	.000
Time 1: start of treatment		Time 2: mid treatment		
Time 3: end of treatment		Time 4: follow up 6 months		
Time 5: follow up 18 months				

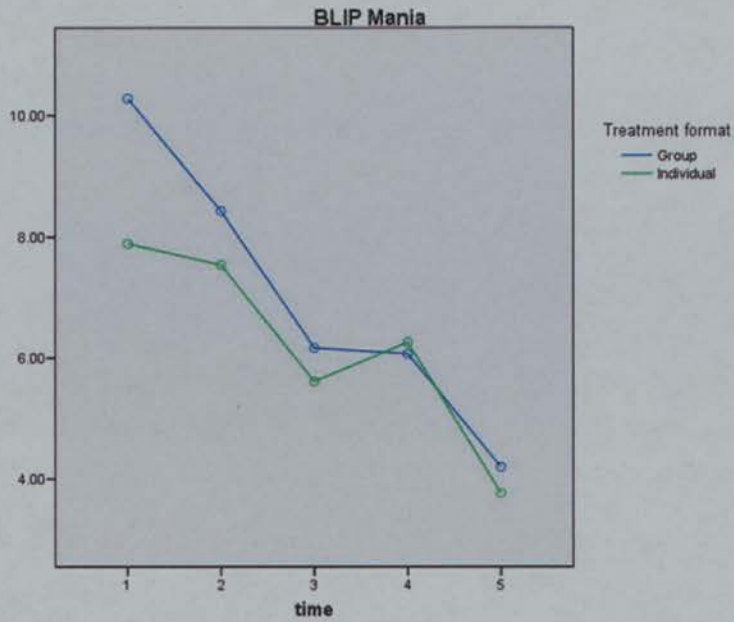


Figure 6.10: BLIP mania scores over time

Table 6.19: Pairwise comparisons of BLIP depression over time points

Time		BLIP D Mean difference	Std. Error	Sig
1	2	2.816*	.914	.002
	3	4.062**	.997	.000
	4	6.286**	1.070	.000
	5	5.942**	1.136	.000
2	3	1.246	.867	.152
	4	3.470**	.830	.000
	5	3.125*	.953	.001
3	4	2.225*	.847	.009
	5	1.880	.987	.058
4	5	-.345	.809	.670

Time 1: start of treatment

Time 2: mid treatment

Time 3: end of treatment

Time 4: follow up 6 months

Time 5: follow up 18 months

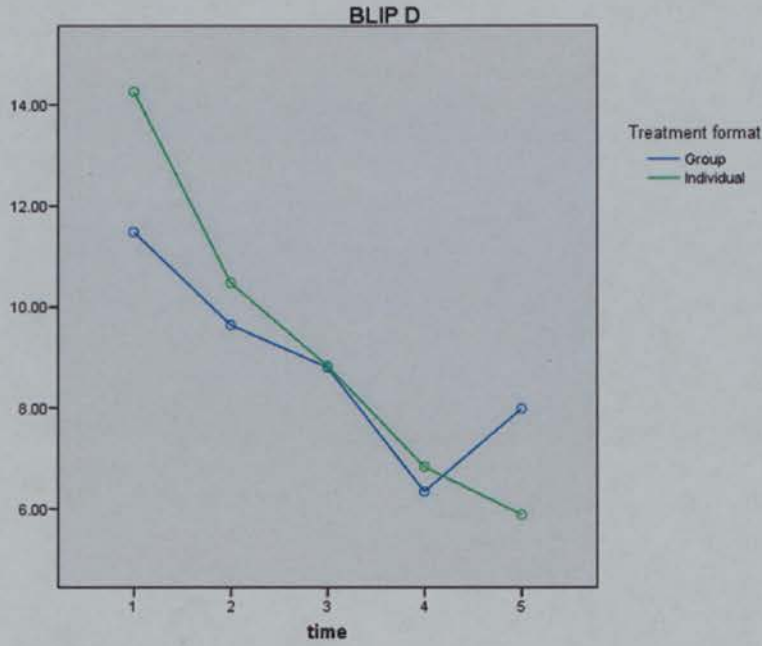


Figure 6.11: BLIP depression scores over time

Table 6.20: Pairwise comparisons of BLIP mixed symptoms over time

Time		BLIP D Mean difference	Std. Error	Sig
1	2	4.023 *	1.591	.012
	3	8.513 **	1.516	.000
	4	8.718 **	1.407	.000
	5	12.290 **	1.580	.000
2	3	4.490 **	1.340	.001
	4	4.695 **	1.304	.000
	5	8.267 **	1.470	.000
3	4	.205	1.125	.855
	5	3.777 *	1.352	.006
4	5	3.572 *	1.127	.002

Time 1: start of treatment	Time 2: mid treatment
Time 3: end of treatment	Time 4: follow up 6 months
Time 5: follow up 18 months	

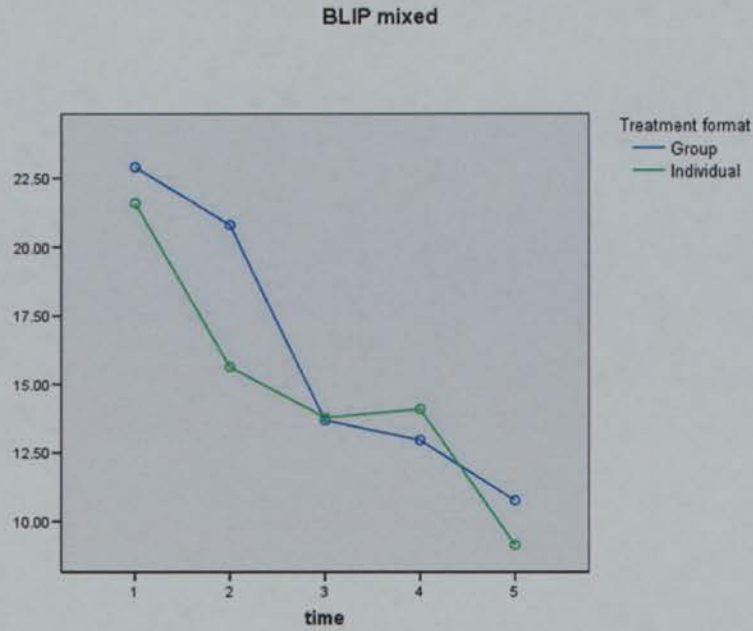


Figure 6.12: BLIP mixed symptoms scores over time

6.4.4 Relapse and recurrence

As outlined above indicators of relapse were approximated using patient information data regarding number of hospital admissions, duration of hospital inpatient stays, and crisis contacts with the psychiatric emergency team as well as recurrence of bipolar disorder mood episodes as determined by the administration of the SCID at the follow up assessments.

Due to the non-normal distribution of the variables relating to number of episodes Friedman's ANOVA's were computed to compare the number and length of admissions between the treatment and the control group. The comparison of the number of bipolar episodes experienced by the waiting list control group (mean=0.83 (sd= 0.77) compared to the psychological treatment group comparing the first six months following treatment (mean=0.33 (sd= 0.40) with the six month TAU condition was significant ($\chi^2(2)=13.01$;

$p = .043$). It was only a small number of participants in each group that experienced an episode during that time period.

Table 6.21 outlines the differences in number and duration of hospital admissions in the 18 months prior to the psychological intervention and during the 18 months following the end of the psychological intervention for treatment group as a whole. Due to the non normal distribution of these variables results of the nonparametric tests are reported.

Table 6.21: Hospital admissions and service contacts pre and post intervention

	Pre treatment Mean (SD)	Post treatment Mean (SD)	Z ^a	Sig
Number of admissions	0.63 (0.75)	0.23 (0.64)	6.34**	.000
Duration of admissions	20.18 (29.47)	7.90 (24.45)	6.87**	.000
No of crisis appointments	1.52 (1.96)	0.22 (0.33)	8.90**	.000

^aWilcoxon Signed Ranks Test

As detailed in the above table participants experienced significantly less hospital admissions, less time in hospital and less psychiatric emergency appointments following the intervention than in a comparable time before.

Significantly more participants (68/ 62.9%) experienced at least one bipolar episode in the 18 months prior to the psychological treatment than in the 18 months following psychological treatment (29/ 26.8%) ($\chi^2(2)=68.72$; $p < .001$). This comparison also applies when the number of depressive episode experienced ($\chi^2(2)=13.01$; $p = .043$) and the number of manic episodes experienced ($\chi^2(2)=6.75$; $p = .034$) are separated.

6.5 Effect of delayed treatment condition on treatment effects over time

When considering the effects of treatment over time on clinical indicators of outcome it is important to consider whether the delayed treatment condition has a main effect on key clinical indicators over time. Therefore, the results of repeated measures ANOVA's and MANOVA's are reported respectively with the main group effect of treatment plus TAU and delayed treatment plus TAU.

In terms of the primary outcome of quality of life, a two way repeated measures MANOVA was performed on the four dimensions of the WHOQOL-BREF. Using Wilks' criterion there was a significant effect of the repeated measures factor, $F(3,189) = 11.43$, $p < .001$, as reported above. There was no significant deviation from parallelism, $F(3,189) = 2.01$, $p = .114$, indicating that the TAU condition of delayed treatment had no effect on the improvement of combined quality of life scores over time.

Secondary outcomes of the relative severity of psychiatric symptoms are considered for observer rated and self report measures. Again, the main effect of repeated measures over time was investigated in interaction with treatment and delayed treatment conditions in order to explore whether any treatment effects in these areas are specific to the delayed treatment condition. Using Wilks' criterion, for the observer rated measures there was no significant interaction effect of time by delayed treatment for the dimensions of the BPRS, $F(5) = 1.27$, $p = .210$; or on the dimensions of the PANSS, $F(5) = 2.01$, $p = .121$.

Equally, for the self report measures, using Wilks' criterion there are no significant interaction effects on the reduction of symptoms over time of delayed treatment when

compared to the treatment group for the depression scores on BDI-II, $F(3,189) = 2.10$, $p = .101$, or the BLIP depression dimension, $F(3,189) = 1.95$, $p = .229$. There is however a significant delayed treatment group by time interaction for the BLIP mania dimension, $F(3,189) = 3.09$, $p = .027$, with individuals in the treatment group showing higher levels of manic symptoms when compared with the delayed treatment group at baseline, but not at end of treatment and the two follow timepoints. The effect of this interaction is small with $\eta_p^2 = 0.04$.

6.6 Summary

At baseline the treatment and control group are comparable in all sociodemographic and clinical characteristics with the exception of length of admission following a hospitalisation in the 18 months prior to treatment being significantly longer in the treatment group than in the control group, possibly indicating a higher level of severity of episodes prior to treatment. There were no significant differences at baseline between men and women or between individuals allocated to the two treatment modalities.

First of all, the main treatment effects were examined using two way repeated measures ANOVA's and two way repeated measures MANOVA's for the key sets of dependent variables relating to the primary outcome variable, quality of life, and to the secondary outcome variables, severity of psychiatric and bipolar symptoms.

In a second set of analyses, for the whole group who received psychological treatment in addition to treatment as usual, changes in quality of life and bipolar symptoms were compared over time, from the baseline assessments to the end of treatment and the follow up periods. Thirdly, treatment effects at the end of treatment and at 18 months follow up were examined for the treatment group as a whole, comparing clinical indicators and indicators of relapse and recurrence over the 18 months prior to the intervention compared with the 18 months following treatment.

In the present study there is a clear positive effect of a formulation based psychological treatment in addition to treatment as usual, psychiatric follow up, when compared to treatment as usual alone.

There is a significant large interaction effect of time by group for the key dimensions of quality of life, indicating that the combined measure of quality of life shows a significantly higher scores for the treatment group when compared to the control group over the same period of time. Very similar results are observed for the secondary outcomes of severity of psychiatric symptoms.

On both observer rated measures, BPRS and PANSS, there are significant large interaction effects of group by time, indicating significant symptom improvement following intervention for the psychological treatment group when compared to treatment as usual.

For the self-report measures of bipolar symptoms there are significant group and time by group interaction effects on measures of depression, (BDI-II), and mania, (Bech Rafealsen Mania Scale), indicating a significant improvement in depression and mania scores following intervention when compared to treatment as usual, with a large effect size for the improvements on these measures. The bipolar specific measure, BLIP, however, showed a significant time effect, but a non significant interaction effect of group by time, on all three bipolar symptom dimensions; manic, depressed and mixed symptoms. This indicates that there was significant improvement over time on the symptom severity as measured by the BLIP, but no significant advantage of psychological intervention plus treatment as usual when compared with treatment as usual alone.

When compared over time individuals in the treatment group as a whole experience a significant reduction in levels of bipolar symptoms, for mania, depression and mixed symptoms as well as a significant improvement in quality of life.

Improvements were evident both on observer rated measures of symptom profile and severity and on self-report measures of subjective levels of distress as well as objective data of service utilisation. These treatment effects were maintained at both follow up periods at six and 18 months following the end of treatment. The only exception to these general results is the changes of scores on the Beck Depression Inventory (BDI-II) and the Bech Rafealsen Mania Scale (BRMS), in that there is a significant increase in scores between the end of treatment and the follow up at 18 months. Even though scores at the final assessment are still significantly lower than at the start of treatment, there is a clear trend that levels of depressive and manic symptoms as assessed by the BDI-II and the BRMS are increasing following treatment.

One possible explanation for this finding is that compared to the more specific measures such as the BLIP, or to the more psychiatric observer rated measures like the PANSS and BPRS, the BDI-II and the BRMS which are frequently employed as screening tools in high risk and normal populations may be more sensitive to subsyndromal levels of emotional distress possibly relating to residual bipolar symptomatology below a diagnosable threshold.

Finally, when compared to the equivalent period of time prior to the psychological intervention the treatment group as a whole appears to have experienced a significant reduction in service utilisation, inpatient admissions and length of inpatient stays in the 18 months following treatment.

VII Cognitive, interpersonal and psychosocial predictors of treatment effects

Which are the key predictors of symptom severity and outcome at the end of psychological therapy and follow up? As outlined in Chapter three two sets of measures relating to psychological and psychosocial factors have been incorporated in the assessments in this trial. The first set of cognitive and psychological factors consists of measures of dysfunctional attitudes (DAS), Personal Beliefs about Illness PBIQ, the Metacognitions Questionnaire (MCQ) and Self Esteem (RSEQ). The second set of interpersonal and psychosocial predictors consists of measures of social support (SOS), perceived expressed emotion (FEICS) and stressful life events and difficulties (LEDS).

It is expected that there will be specific differential effects of cognitive and interpersonal factors regarding outcome and therapeutic change as the integrated treatment is aimed at both cognitive and interpersonal aspects of individual participants' functioning. Further, a number of specific hypotheses regarding the influence of cognitive, psychological and psychosocial variables have been put forward in recent years; in particular regarding the effects of dysfunctional attitudes, self esteem, and life events in relation to mania and depression in individuals with bipolar disorder.

In this chapter I aim to address the question from two perspectives; (I) are psychological and psychosocial characteristics at baseline predictive of key clinical characteristics; and

(2) are psychological and psychosocial factors predictive of outcome at end of treatment and follow up.

7.1 Psychological and psychosocial sample characteristics at baseline

7.1.1 Cognitive and psychological variables

The key cognitive and psychological variables are outlined in Table 7.1. The examination of the pattern of distribution through histograms and Kolmogoroff-Smirnoff test statistics of these variables showed that all variables in this group are sufficiently normally distributed. All psychological and psychosocial variables have been systematically compared between the treatment and TAU group, gender and by treatment modality. The differences between treatment and TAU groups are used to illustrate the sample characteristics for these variables.

Table 7.1: Psychological characteristics by treatment group

	Treatment Group (n=106)	Control Group (n=96)
	Mean (sd)	Mean (sd)
Personal Beliefs about Illness Questionnaire (PBIQ)		
Loss	10.1 (2.7)	9.7 (2.4)
Humiliation	6.6 (2.1)	6.7 (2.0)
Shame	7.2 (1.9)	7.0 (1.9)
Entrapment	4.0 (1.1)	4.0 (1.2)
Self versus Illness	9.4 (2.6)	9.8 (2.7)
Metacognition Scale (MCQ)		
Positive Beliefs	1.6 (0.4)	1.7 (0.4)
Uncontrollability	2.6 (0.7)	2.4 (0.7)
Cognitive Confidence	2.2 (0.7)	1.9 (0.6)
Negative thoughts	2.0 (0.6)	1.9 (0.6)
Self consciousness	2.5 (0.7)	2.2 (0.7)
Dysfunctional Attitude Scale (DAS)		
Achievement	28.6 (11.8)	30.0 (11.5)
Dependency	29.8 (8.3)	31.4 (10.2)
Self-control	28.2 (8.3)	29.0 (7.5)
Rosenberg Self Esteem Scale (RSES)	23.4 (5.9)	24.9 (6.4)

As detailed in Table 7.1, there are no significant differences between the two groups in this set of measures. There are also no significant differences in these variables between individuals in the two treatment modalities. There are however some significant differences between male and female participants; in that women show a significantly higher level of 'Entrapment' ($t(189)=-2.42$; $p=.016$) and 'Shame' ($t(189)=-2.28$; $p=.011$) on the PBIQ, and higher scores on the DAS 'Depression' ($t(189)=-2.98$; $p=.003$) and 'Achievement' ($t(189)=-2.88$; $p=.005$) dimensions.

Consequently in the subsequent regression analyses gender will be taken into account as a covariate.

7.1.2 Psychosocial variables

Table 7.2 details the differences in the psychosocial variables, social support (SOS), perceived expressed emotion (FEICS) and life events and difficulties (LEDS) between the two treatment groups at baseline. The examination of the pattern of distribution through histograms and Kolmogoroff-Smirnoff test statistics of these variables showed that with exception of the LEDS all variables in this group are sufficiently normally distributed. Differences between treatment modality and gender at start of treatment were also analysed.

Table 7.2: Psychosocial characteristics by treatment group

	Treatment Group (n=106)	Control Group (n=96)
	Mean (sd)	Mean (sd)
Significant Others Scale (SOS)		
Emotional Support	5.0 (0.9)	5.1 (1.0)
Practical Support	4.7 (0.8)	4.5 (1.0)
Support Total	4.6 (0.9)	4.5 (0.9)
Dissatisfaction Emotional S	1.1 (0.8)	1.0 (0.8)
Support Dissatisfaction	2.2 (1.3)	1.9 (1.4)
Family Emotional Involvement and Criticism Scale (FEICS)		
Total	2.5 (0.4)	2.6 (0.5)
Criticism	2.5 (0.6)	2.4 (0.7)
Emotional Involvement	2.6 (0.5)	2.7 (0.5)
Life Events and Difficulties Schedule (LEDS)		
Independent negative LE	3.8 (0.3)	3.2 (0.3)
Independent positive LE	1.2 (0.1)	1.4 (0.1)
Chronic difficulties	3.9 (0.2)	3.7 (0.2)

As outlined in Table 7.2 there are no significant differences between these two groups in respect of social support, expressed emotions and life events and difficulties. Comparison between treatment modalities at start of treatment also revealed no significant differences in this set of measures. The comparison of the gender groups at

the start of treatment however showed that women have significantly higher scores on perceived 'Emotional Involvement' ($t(124)=-2.68$; $p=.008$) and overall EE ($t(124)=-2.42$; $p=.017$) on the FEICS. There are no significant differences between male and female participants in these measures.

Also, age and time since the initial diagnosis of bipolar disorder were not significantly correlated with any of the above mentioned psychological and psychosocial variables.

7.2 Changes in psychological and psychosocial characteristics following treatment

As a next step in the analysis of the cognitive, interpersonal and psychosocial factors in this trial the impact of psychological treatment on these variables was examined utilising repeated measures ANOVA's.

7.2.1 Psychological variables

Tables 7.3 illustrates the within subject differences for the cognitive and psychological variables over the assessed time points.

Table 7.3: Repeated measures ANOVA's for psychological variables

Variable within subject effects		F (df); sig Time	F (df); sig Time*TreatmentMode
Personal Beliefs about Illness Questionnaire (PBIQ)	Loss	39.78** (4,188); p< .001	0.68 (4,188); p= .408
	Humiliation	35.77** (4,188); p< .001	0.24 (4,188); p= .913
	Shame	7.97** (4,188); p< .001	0.21 (4,188); p= .872
	Entrapment	32.58** (4,188); p< .001	1.79 (4,188); p= .129
	Self versus Illness	17.62** (4,188); p< .001	0.19 (4,188); p= .851
Metacognition Scale (MCQ)	Positive Beliefs	4.93* (3,189); p= .002*	2.47 (3,189); p= .062*
	Uncontrollability	15.54** (3,189); p< .001	0.60 (3,189); p= .606
	Cognitive Confidence	9.23** (3,189); p< .001	0.10 (3,189); p= .999
	Negative thoughts	20.78** (3,189); p< .001*	1.15 (3,189); p= .326*
	Self consciousness	9.07* (3,189); p= .003*	0.67 (3,189); p= .570*
Dysfunctional Attitude Scale (DAS)	Achievement	11.88** (3,189); p< .001	1.01 (3,189); p= .387
	Dependency	7.54** (3,189); p< .001	0.72 (3,189); p= .538
	Self-control	15.23** (3,189); p< .001	0.51 (3,189); p= .671
Rosenberg Self Esteem Scale (RSES)		21.07** (3,189); p< .001	0.76 (3,189); p= .515

*Sphericity not assumed (Greenhouse-Geisser correction applied)

As can be seen in Table 7.3 all cognitive and psychological variables change significantly over time. Generally the pattern of change is very similar across the three measures in that there is a clear observable and significant improvement in scores over the course of treatment, which is generally maintained through the follow up period. There is no significant interaction effect with treatment modality for any of these variables.

There are two exceptions to the DAS 'dependency' score where a clear reduction is observable to the end of treatment which is maintained at six months follow up but then increases significantly at 18 months follow up, the final score is still significantly lower than at start of treatment (p= .033). The Rosenberg Self Esteem measure also shows a

marked different pattern of change over time, in that there is a significant dip in the overall self esteem score at the end of treatment which returns at follow up o a comparable level to the baseline scores; the final score is significantly higher than at baseline ($p = .012$).

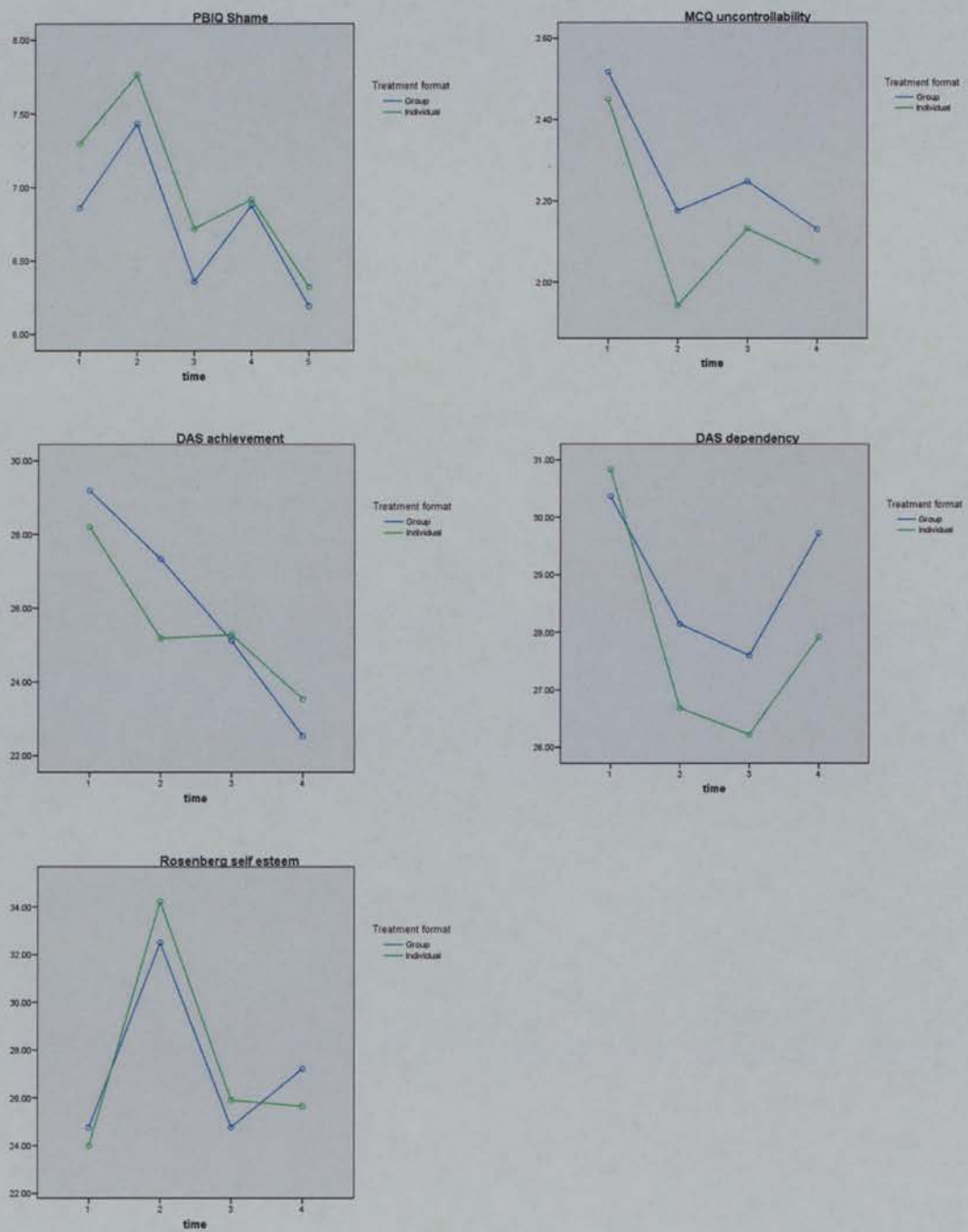


Figure 7.1: Change in predictor variables over time

7.2.2 Psychosocial variables

Table 7.4 outlines the changes over the assessment time points for the interpersonal variables social support and expressed emotion.

Table 7.4: Repeated measures ANOVA's for psychosocial variables

Variable within subject effects	F (df); sig Time	F (df); sig Time*TreatmentMode
Significant Others Scale (SOS)		
Emotional Support	.09 (3,189); $p = .964^*$	2.16 (3,189); $p = .091^*$
Practical Support	0.79 (3,189); $p = .499$	1.70 (3,189); $p = .144$
Support Total	0.88 (3,189); $p = .496$	7.76* (3,189); $p < .006$
Dissatisfaction Emotional S	3.18* (3,189); $p = .024^*$	1.42 (3,189); $p = .221$
Support Dissatisfaction	2.34 (3,189); $p = .072^*$	0.53 (3,189); $p = .661^*$
Family Emotional Involvement and Criticism Scale (FEICS)		
Total	7.76** (3,189); $p < .001^*$	0.35 (3,189); $p = .152^*$
Criticism	9.30** (3,189); $p = .002$	1.68 (3,189); $p = .169$
Emotional Involvement	5.76* (3,189); $p = .001^*$	1.20 (3,189); $p = .306^*$

*Sphericity not assumed (Greenhouse-Geisser correction applied)

The pattern of change as illustrated in Graph 7.2 is mixed for this group of variables; there are significant treatment effects for individual's dissatisfaction with the emotional support from close others reducing over time ($F(3,189)=3.18^*$; $p = .024$) and generally their level of perceived expressed emotion reducing over the course of treatment and follow up at six months and then increasing again at the follow up at 18 months ($F(3,189)=7.76$; $p < .001$).

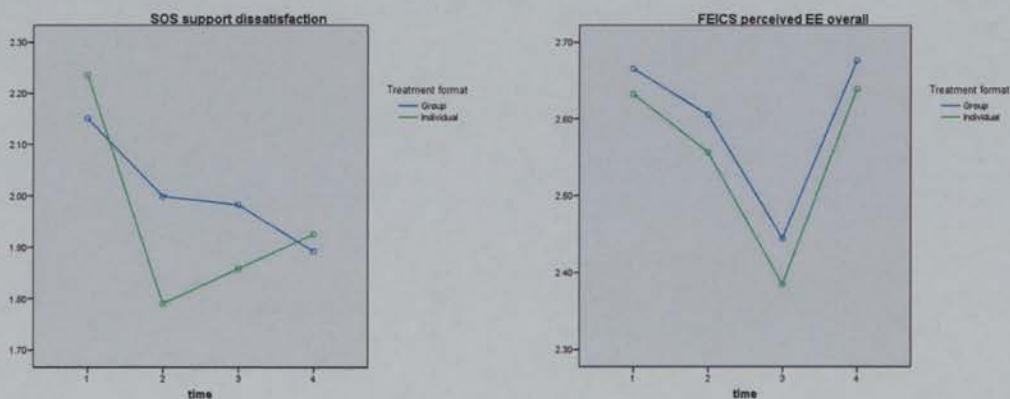


Figure 7.2 Changes in perception of social support

Secondly, as illustrated in Graph 7.3 we can observe a clear interaction effect with treatment modality in that individuals' who attended the groups show a significant decrease in their social support score relating to their significant others which was maintained at follow up, and individuals who received individual treatment showed a significant increase in their perceived support from close others which was also maintained at follow up ($F(3,189)=7.76$; $p<.006$).

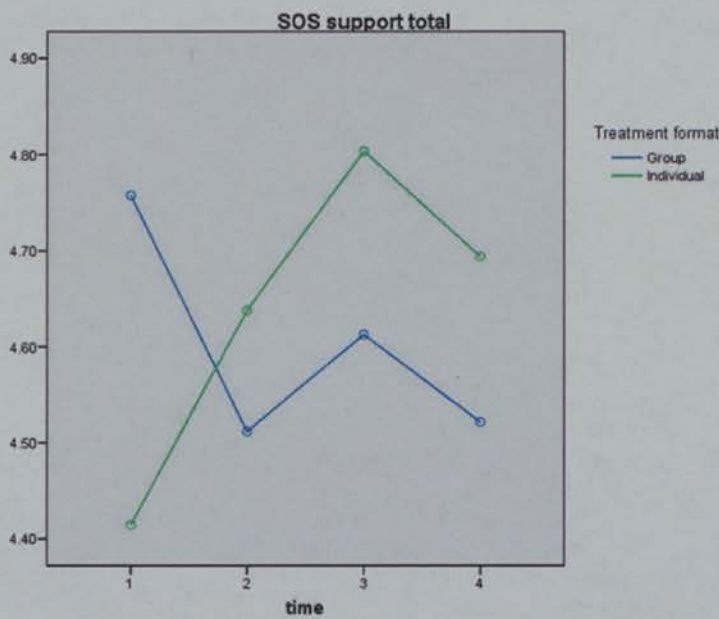


Figure 7.3 Changes in overall social support

7.3 Statistical Analysis

A series of multiple linear regression and logistic regression models were calculated to determine the main predictors associated with clinical characteristics in the study samples at baseline. A second set of multiple linear regression models was constructed to determine the main psychological and psychosocial predictors of the key outcome variables at end of treatment and follow up. Prior to these analyses test for the normal

distribution, extreme outliers, collinearity and multicollinearity, as well as homoscedasticity were carried out for the relevant predictor variables. As outlined above with the exception of life events and difficulties no extreme outliers were found and all independent variables were sufficiently normally distributed.

The predictor variables were entered hierarchically and by forced entry in each hierarchical block to avoid purely statistical biases caused by a stepwise or preferred entry. Factors that are known to have a significant bearing on level of distress and symptomatology in bipolar disorder such as self esteem or significant life events and stressors are entered first after a set of identified covariates (duration between estimated onset and diagnosis, number of previous episodes and time since original diagnosis); all other relevant cognitive and psychosocial factors are entered in subsequent sets. Detailed results are reported for the significant predictors of symptomatology, indicators of relapse and recurrence and quality of life.

The dependent variables for bipolar symptomatology are naturally highly correlated and in order to prevent repetitive and multiple regression models being tested on sets of closely related dependent variables thus inflating the alpha error and devaluing the validity of the models tested a factor analysis (maximum likelihood) was carried out on all symptom and distress related dependent variables. From this analysis three clear factors emerged explaining 73.7% of the variance and accommodating all relevant scales and subscales. The three factors are: (1) General psychiatric symptoms, including any positive and negative symptom dimensions, (2) Depression, and (3) Mania. The factor loadings of the related scales are detailed in Table 7.5. The three factors further seem sufficiently independent and show no significant intercorrelation as detailed in Table 7.6.

Table 7.5: Factor loadings on three dimensions of bipolar symptoms

	General	Factor Depression	Mania
BPRS	.946		
BPRS depression	.790	.392	
PANSS general	.733		
BPRS negative symptoms	.600		
PANSS positive symptoms	.502		
PANSS negative symptoms	.471		
BDI-II		.822	
BLIP depression		.644	
BRMS			.702
BLIP mania			.679
AMS			.512

Table 7.6: Pearson's correlations of distress dimensions

	General	Factor Depression	Mania
General		.055	.042
Depression	.055		.061
Mania	.042	.061	

In the subsequent multiple regression analyses the three factors will be used as dependent variables as well as quality of life and indicators of relapse and recurrence for the predictors of outcome analysis.

7.4 Baseline predictor models

7.4.1 Predictors of bipolar symptoms at baseline

Table 7.8 outlines the regression models for the three main symptom dimensions at baseline. For these multiple regression models the first set of predictors consisted of

gender, age of diagnosis, number of previous episodes and number of hospital admissions; the second set consisted of childhood trauma, and the LEDS variables; the third set was made up from self esteem and cognitive factors (DAS, PBIQ, MCQ); and finally in the last set the psychosocial variables of social support (SOS) and expressed emotion (FEICS) were added.

Table 7.8: Multiple regression analyses and predictors for symptom dimensions

Variable	mult R	F (df)	sig. F	Beta	T	sig. T
General psychiatric symptomatology	.402*	2.24 (3,132)	p= .043			
LEDS, number of negative LE				.227*	2.22	p= .022
LEDS, number of positive LE				-.197*	-2.32	p= .028
Depression	.769**	14.28 (14,132)	p< .001			
LEDS, number of positive LE				-.250*	-3.06	p= .003
LEDS, number of negative LE				.264*	2.69	p= .008
Rosenberg self esteem				-.632**	-8.96	p< .001
MCQ cognitive confidence				.235*	3.16	p= .002
PBIQ shame				.159*	2.16	p=.032
Mania	.678**	8.25 (3,132)	p< .001			
Age at diagnosis				-.225*	3.23	p= .002
Childhood trauma				.151*	2.01	p= .047
LEDS, number of positive LE				.194*	2.44	p= .016
LEDS, number of negative LE				.227*	2.47	p= .015
MCQ, cognitive confidence				.268*	3.14	p= .002
MCQ, negative thoughts				.252*	2.83	p= .005

As illustrated in Table 7.8 all three dimensions of baseline bipolar symptomatology have strong predictor variables explaining between 16 and 59% of variance. The only common predictors to all three dimensions are significant negative and positive life events as measured by the LEDS. In addition to these predictors the depression dimension is

further determined by self esteem, cognitive confidence and shame. The mania dimension on the other hand is explained by two further historical variables, age of diagnosis and childhood trauma, as well as the meta cognitive factors of cognitive confidence and negative thoughts. None of the psychosocial variables of social support and expressed emotion helped to explain level of symptomatology at baseline once the outlined predictors have been taken into account.

7.4.2 Predictors of Quality of Life at baseline

In table 7.9 the regression models for the four quality of life dimensions are illustrated. The same hierarchical sets of predictors were entered as above, with the discrepancy that cognitive and psychosocial variables were entered in the same set of predictors as less specific hypotheses regarding quality of life are put forward.

Table 7.9: Multiple regression analyses and predictors for quality of life (whoqol-bref) – at baseline

Variable	mult R	F (df)	sig. F	Beta	T	sig. T
Physical well being	.723**	68.6 (2,135)	p< .001			
Rosenberg self esteem	.330*			.330*	2.57	p= .012
PBIQ, loss	.236*			.236*	2.20	p= .030
PBIQ, humiliation	-.393**			-.393**	-3.82	p< .001
PBIQ, shame	-.237*			-.237*	-2.48	p= .014
Psychological well being	.820**	74.45 (2,135)	p< .001			
Rosenberg self esteem	.296*			.296*	2.75	p= .007
PBIQ, humiliation	-.260*			-.260*	-2.90	p= .004
PBIQ, shame	-.192*			-.192*	-2.37	p= .019
MCQ cognitive confidence	-.197*			-.197*	-2.46	p= .015
Social relationships	.579**	20.32 (2,135)	p< .001			
Rosenberg self esteem	.250*			.250*	2.75	p= .007
PBIQ, shame	-.249*			-.249*	-2.21	p= .029
SOS, emotional support	-.519*			-.519*	-2.10	p= .038
SOS, support total	-.803*			-.803*	2.27	p= .028
Environment	.761**	37.62 (2,135)	p< .001			
LEDS, number of negative LE	-.218*			-.218*	-2.63	p= .010
MCQ, cognitive confidence	-.358**			-.358**	-4.00	p< .001
DAS, dependency	.222*			.222*	2.19	p= .030
SOS, support dissatisfaction	-.539*			-.539*	-2.83	p= .006
SOS, emotional support dissatisfaction	.450*			.450*	2.09	p= .039
FEICS, perceived criticism	.294**			.294**	3.27	p< .001

For the first two quality of life dimensions with self referencing items, namely physical and psychological well being, self esteem, loss, humiliation and shame are key predictors; for the social relationships dimension social support, self esteem and shame are highly predictive, whereas for environmental well being, a mixture of interpersonal factors,

cognitive confidence and negative life events are predictive of altogether 58% of the variance of this domain. Altogether between 34 and 67% of variance in the quality of life dimensions at baseline are explained by this small set of psychological and psychosocial predictors.

It seems noteworthy that the set of predictors explaining quality of life as a generic indicator of satisfaction and emotional well being is notably different from the set of variables explaining the majority of variance in the specifically symptom related factors.

7.5 Predictors of outcome

In a second set of multiple regression analyses the cognitive, interpersonal and psychosocial predictors of outcome variables were examined. Predictors were considered in the same hierarchical steps as for the predictor analyses at baseline; in addition symptom levels at start of treatment were entered as the first step in the hierarchical multiple regression analyses. All multiple regression analyses were performed for outcomes at end of treatment and 18 months follow up.

Maximum Likelihood factor analyses were carried out for symptom scores at end of treatment and 18 months follow up resulting in the same three symptom dimensions: general psychiatric symptoms, Depression and Mania. Tables 7.10 and 7.11 outline the factor loadings at end of treatment and 18 month follow up. The factors at either of these time points were not significantly correlated and could be used independently in the multiple regression analyses.

Table 7.10: Factor loadings on three dimensions of bipolar symptoms at end of treatment

	General	Factor Depression	Mania
BPRS	.952		
BPRS depression	.764		
PANSS general	.755	.368	
BPRS negative symptoms	.641		
PANSS negative symptoms		.343	
BDI-II		.687	
BLIP depression		.693	
BRMS			.869
BLIP mania			.746
AMS			.742

Table 7.11: Factor loadings on three dimensions of bipolar symptoms at follow up

	General	Factor Depression	Mania
BPRS	.774		
BPRS depression	.693		
PANSS general	.688		
BPRS negative symptoms	.607		
PANSS negative symptoms	.368		
BDI-II		.352	
BLIP depression		.752	
BRMS			.667
BLIP mania			.701
AMS			.703

7.5.1 Predictors of bipolar symptoms following treatment

Table 7.12: Multiple regression analyses and predictors for bipolar symptom dimensions at end of treatment

Variable	mult R	F (df)	sig. F	Beta	T	sig. T
General psychiatric symptomatology	.580*	2.44 (8,184)	p= .003			
PBIQ, shame				.225*	2.74	p= .007
MCQ, cognitive confidence				.256*	2.94	p= .004
SOS, practical support				-.234*	-2.03	p= .044
SOS, support overall				.389*	3.38	p= .001
Depression	.749**	10.34 (8,184)	p< .001			
Depression at baseline				.124*	2.30	p= .022
Mania at baseline				-.153*	-2.93	p= .003
PBIQ, loss				.361**	4.84	p< .001
PBIQ, humiliation				.217**	3.22	p< .001
PBIQ, shame				.189*	2.83	p= .005
PBIQ, entrapment				-.181*	-2.78	p= .006
MCQ, uncontrollability				.188*	2.64	p= .009
SOS, support overall				-.352**	-3.76	p< .001
Mania	.747**	3.73 (8,184)	p< .001			
MCQ, positive beliefs				.196*	2.77	p= .006
MCQ, cognitive confidence				.181*	2.53	p= .012
MCQ, self consciousness				.275**	4.12	p< .001
DAS, self control				.366**	5.93	p< .001
FEICS, criticism				.573**	6.83	p< .001

As illustrated in Table 7.12 largely cognitive and interpersonal variables at baseline explain between 33 and 56% of variance of the symptom scores at the end of treatment. In these regression models it appears that depression at the end of treatment is mainly associated with negative appraisals of illness and social support, whereas mania appears to be related to positive self related beliefs and level of criticism from others. It is noteworthy that in

all hierarchical multiple regressions that relate to symptom dimensions as outcome variables levels of symptoms at baseline were highly predictive before any of the psychological and psychosocial variables were added to the regression models but that their predictive power was completely subsumed or significantly reduced by these variables in the final model.

The parallel regression models for the dimensions of bipolar symptoms at 18 month follow up reveal a very similar pattern. Overall between 34 and 44% of variance in symptom scores at follow up are explained by the model.

The main predictors for depression ($R = .614$; $F(8,184)=4.98$; $p < .001$) at follow up are: LEDS, number of negative life events ($\beta = .179$; $t=2.61$; $p = .010$); PBIQ, loss ($\beta = .346$; $t=4.21$; $p < .001$); PBIQ, shame ($\beta = -.194$; $t=-2.41$; $p = .017$); MCQ, negative beliefs ($\beta = .188$; $t=2.37$; $p = .019$); DAS, dependency ($\beta = -.265$; $t=-3.39$; $p = .001$); and SOS, dissatisfaction with emotional support ($\beta = -.234$; $t=-2.92$; $p = .004$).

The main predictor variables for the mania dimension ($R = .583$; $F(8,184)=2.66$; $p < .001$) at follow up are: PBIQ, entrapment ($\beta = .186$; $t=2.36$; $p = .019$); MCQ, positive beliefs ($\beta = -.166$; $t=-2.82$; $p = .028$); MCQ, self consciousness ($\beta = .195$; $t=2.73$; $p = .007$); DAS, self control ($\beta = .192$; $t=2.59$; $p = .010$).

7.5.2 Predictors of quality of life following treatment

Table 7.13 shows the significant predictors for quality of life at the end of treatment. Hierarchical multiple regression models were calculated in parallel to the multiple regressions reported in relation to symptom related outcomes.

Table 7.13: Multiple regression analyses and predictors for quality of life (whoqol-bref) – at outcome

Variable	mult R	F (df)	sig. F	Beta	T	sig. T
Physical well being	.660**	46.21 (8,183)	p< .001			
LEDS, number of negative life events				.144*	2.65	p= .025
PBIQ, humiliation				-.158*	-2.08	p= .038
PBIQ, shame				-.194*	-2.72	p= .007
DAS, achievement				-.310**	-3.52	p< .001
DAS, self control				.135*	2.12	p= .035
Psychological well being	.839**	45.69 (8,183)	p< .001			
General psychiatric symptoms at baseline				-.128*	-2.89	p= .004
LEDS, number of negative LE				.146	2.95	p= .004
PBIQ, loss				-.176*	-2.69	p= .008
PBIQ, humiliation				-.215**	-3.87	p< .001
PBIQ, shame				-.161*	-2.93	p= .004
SOS, support total				-.240*	-2.93	p= .004
SOS, support dissatisfaction				-.345**	-5.03	p< .001
FEICS, total score				.179*	2.45	p= .015
Social relationships	.735**	39.45 (8,183)	p< .001			
LEDS, number of negative LE				-.192**	-3.31	p= .001
MCQ, positive beliefs				-.222*	-3.05	p= .003
MCQ, negative thoughts				.192*	2.43	p= .016
DAS, achievement				-.339**	-3.77	p< .001
SOS, emotional support				-.298*	-3.28	p= .001
SOS, emotional support dissatisfaction				-.171*	-1.99	p= .047
FEICS, criticism				.190*	2.20	p= .029
FEICS, total				-.237*	-2.60	p= .010
Environment	.627**	33.82 (8,183)	p< .001			
PBIQ, entrapment				.170*	2.34	p= .020
DAS, achievement				-.278**	-3.02	p< .001
DAS, dependency				.220*	2.39	p= .018
FEICS, perceived criticism				.217*	2.26	p= .025

Between 3 and 70% of variance of the quality of life scores at end of treatment are explained by the hierarchical multiple regression models outlined in Table 7.13. The predictors of psychological well being at the end of treatment are similar to those observed for the depression dimension, baseline symptoms, negative life events and negative cognitions such as thoughts relating to loss, humiliation and shame as well as significant interpersonal factors such as support satisfaction and the level of perceived expressed emotion.

At 18 months follow up between 30 and 48% of variance in quality of life scores can be explained by these regression models. Key predictors psychological well being at follow up ($R = .699$; $F(8,133)=4.98$; $p < .001$) are the interpersonal variables: SOS, emotional support ($\beta = -.351$ $t = -3.92$; $p < .001$); SOS support dissatisfaction ($\beta = -.306$ $t = -3.45$; $p = .001$); FEICS, criticism ($\beta = .180$ $t = 2.30$; $p = .023$); and the cognitive factors: PBIQ, shame ($\beta = -.278$ $t = -3.07$; $p = .003$); PBIQ, entrapment ($\beta = .241$ $t = 2.95$; $p = .004$); and MCQ, uncontrollability ($\beta = -.415$ $t = -4.41$; $p < .001$).

The key predictors for the social relationships dimension of the quality of life measure ($R = .641$; $F(8,133)=11.97$; $p < .001$) at 18 months follow up are: SOS, support total ($\beta = .263$ $t = 2.49$; $p = .014$); FEICS, criticism ($\beta = -.185$ $t = -2.21$; $p = .029$); DAS, dependency ($\beta = -.218$ $t = -2.34$; $p = .021$); DAS, self control ($\beta = -.182$ $t = -2.03$; $p = .044$); and MCQ, positive beliefs ($\beta = .204$ $t = 2.17$; $p = .031$).

7.5.3 Predictors of relapse and recurrence

As a last step in the investigation of baseline psychological and psychosocial variables association with clinical outcomes following treatment logistic regression models were

calculated to determine predictors for relapse and recurrence at 18 months follow up. For the purposes of this analysis relapse has been defined by any depressive or manic episode experienced during the follow up period as identified by the SCID. The same variables were entered in hierarchical sets as for the linear multiple regression models above. Three main dependent variables were defined for this analysis: (1) Occurrence of any bipolar episode during the follow up period; (2) occurrence of a depressive episode during the follow up period; and (3) occurrence of a manic episode during the follow up period. Table 7.14 shows the overall model significance (χ^2), the log likelihood of observed and predicted values over probabilities of observed and predicted values, R^2 equivalence (Nagel Vierkes), and significant predictors for each model.

Table 7.14: Logistic regression analyses and predictors for relapse at 18 month follow up

Variable	χ^2 / Log Likelihood	R^2 equiv	(df)	sig.	B	Wald χ^2	sig.
Bipolar episode	8.96/ 253.76	.153	8	p= .345 p= .112			
Episode of depression	8.96/ 258.71	.179	8	p= .345 p= .133			
					LEDS, number of negative LE	.305*	5.22 p= .022
Episode of mania	17.44/ 284.24	.241	8	p= .032 p= .046			
					Depressive symptoms at baseline	-.394*	4.06 p= .044
					LEDS, number of negative LE	.117*	3.98 p= .046
					MCQ, uncontrollability	.985*	9.11 p= .003
					FEICS, total	.692*	4.93 p= .026

As detailed in Table 7.14 the hierarchical logistic regression analysis does not yield a satisfactory regression model for relapse overall or depression relapse. Significant predictors for a manic relapse are depressive symptoms at baseline, negative life events, expressed emotion and meta-cognitions relating to uncontrollability of own thoughts. Multiple regressions were further calculated for other indicators of relapse such as number of hospital admissions and length of inpatient stays, but similarly these regressions did not yield overall significant predictor models.

7.6 Summary

The exploration of cognitive, interpersonal and psychosocial variables for in a clinical sample of bipolar disorder in this study has been addressed on two levels. First, in an analysis of predictors of profile and severity of bipolar symptoms in a series of cross sectional analyses; and second, in the investigation of psychological and psychosocial predictors of outcomes and treatment effects following the trial of psychological intervention.

As any psychological intervention the treatment implemented in this trial is designed not only to intervene at a level of symptomatic distress but to target psychological variables that may mediate and influence a range of factors associated with reduced symptoms, emotional recovery and productive coping. The key elements of this intervention specifically focused on metacognitive awareness and reflective capacity to enhance affect regulation and on perceived difficulties and dissatisfactions in the close social networks of participants. These target areas were attempted to be assessed using a range of psychological measures.

These underlying questions were addressed in three steps. First, a systematic comparison of key cognitive, interpersonal and psychosocial variables over time was carried out in order to assess whether these psychological factors changed significantly through psychological intervention and whether these potential changes could be maintained over a period of time. Second, the association of these variables on the level of bipolar symptoms and quality of life was explored at baseline to determine possible patterns of predictive association with specific bipolar symptom dimensions and quality of life. Third, the prediction of outcome was analysed in an analogue framework to the

analyses at baseline to determine which psychological indicators predicted treatment effects at outcome and follow up.

The first set of repeated measures ANOVA's determined that there are significant changes in all areas of assessed cognitive, interpersonal and psychosocial variables. These findings confirm a clear intervention effect on underlying psychological characteristics in this group of bipolar patients, with the exception of levels of perceived social support where there is a significant change in the key variable of support satisfaction for the individuals attending the group treatments only; and in the measure of expressed emotion where there is only a significant improvement over the course of treatment that could not be maintained at follow up. Possible explanation for these exceptions could be that especially in terms of the systemic factors of expressed emotion in close social networks a non-systemic intervention that only targets the individual patients cannot achieve lasting change in the social environment of the individuals.

In terms of the main predictors of symptomatic distress and quality of life in this sample of bipolar patients it appears that there is an emerging pattern of associated predictors regarding different symptom dimensions and quality of life. It seems that in the multiple regression analyses general psychiatric symptomatology is solely associated with significant negative life events, whereas the two dimensions of affective symptoms, levels of depression and mania, are significantly associated with negative life events and key cognitive factors relating to cognitive confidence, self esteem and shame for depressive symptoms and cognitive confidence and negative metacognitive beliefs for manic symptoms. The health related dimensions of quality of life at baseline appear to be significantly associated with self esteem and beliefs regarding humiliation, shame; whereas the social and environmental dimensions of quality of life appear to be predicted by

levels of social support and cognitions relating to interpersonal functioning. This is possibly opening up a level of functioning in bipolar disorder that may be independent of symptomatic distress and that may be susceptible to change through psychological intervention.

In the analyses relating to the prediction of key outcome indicators it appears that there is a different pattern of variables impacting on the dimensions of bipolar symptoms and quality of life at the end of treatment and follow up compared with the significant psychological and psychosocial predictors at baseline. Significant negative life events no longer have a strong association with levels of bipolar symptoms and there are more significant interpersonal variables as well as cognitive characteristics. For the general psychiatric symptoms main predictors are shame, cognitive confidence and levels of social support. Depression is strongly associated with loss, humiliation and shame as well as symptoms at baseline and beliefs about controllability. Manic symptoms are predicted by positive metacognitive beliefs, cognitive confidence and self control as well as the criticism dimension of expressed emotion.

Quality of life following treatment is significantly related to negative life events, shame, loss and dysfunctional attitudes relating to achievement and self control for the health related dimensions of quality of life. The environmental and social dimensions of quality of life are predicted by a mixture of negative metacognitive characteristics, dysfunctional attitudes regarding achievement and dependency as well as interpersonal factors, such as criticism and dissatisfaction with social support.

In terms of relapse and recurrence it seem to be only negative life events, for the recurrence of depressive episodes; and depression at baseline as well as negative life

events, metacognitive beliefs about uncontrollability and expressed emotion for manic episodes that have a significant predictive effect.

Overall the hierarchical multiple regression models reported in this section reach a high level of explanatory power with between 35% and 70% of the total variance of the dependent variables being explained by the models.

VIII Structural models of treatment effects

In chapter 7 a series of multiple regression analyses were performed to investigate the predictors of level of symptomatic distress at baseline and to analyse psychological and psychosocial predictors of key outcome variables at end of treatment and follow up. Predictor variables were drawn from two sets of measures; cognitive and psychological factors and interpersonal and psychosocial predictors.

Multivariate regression models are limited in that they only permit parallel testing of a set of indicators of outcome, or dependent variables and there is limited scope to test specific hypotheses of interaction effects between sets of independent variables. It is expected that there will be specific differential effects of cognitive and interpersonal factors regarding outcome and therapeutic change as the integrated treatment is aimed at both cognitive and interpersonal aspects of individual participants' functioning and a main effect of level of severity of the disorder and independent life events on outcome. In order to test specific mediating effects of cognitive and interpersonal factors taking into account the level of symptomatic distress at the start of treatment as well as the influence of independent life events and stressors a series of covariance models was carried out using structural equation modelling techniques (SEM).

8.1 Hypotheses

As part of the testing of Hypothesis 3, that changes in psychosocial, cognitive and emotional factors will be predictive of improved quality of life, reduced severity of symptoms and reduced indicators of relapse, structural and longitudinal Covariance modelling (SEM) was utilised to investigate two specific questions:

- A) There will be specific and differentiating interacting and mediating effects of cognitive, interpersonal and psychosocial variables relating to levels of depression and mania at baseline.
- B) There will be a full meditational effect of cognitive, interpersonal and psychosocial variables on outcome for levels of depression and mania.

A series of a priori path models have been constructed and evaluated for overall model fit and specific path indices. To illustrate the models tested Figures 8.1 and 8.2 offer a schematic outline of the basic models tested at baseline and over time.

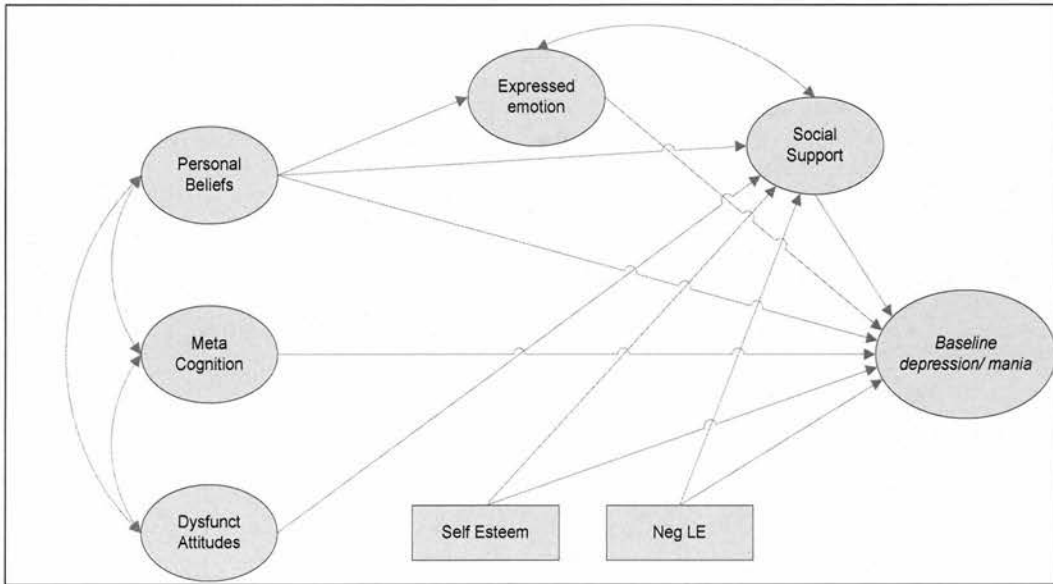


Figure 8.1: Schematic baseline mediation model for depression and mania

As detailed in the schematic illustration in Figure 8.1 at baseline a series of parallel direct and mediating effects of key psychological and psychosocial variables are being tested in a structural model of key predictors and indicators of general psychiatric symptomatology, depression and mania respectively. The hypothesised direct and mediating effects are based on theoretical assumptions of the direct effects of self esteem and life stressors on level of symptoms and the interacting effects of cognitive and interpersonal characteristics, such as dysfunctional attitudes and personal beliefs about illness being dependent on other appraisal, perceived support and family environment. The most basic version of the model represents the multiple regressions reported in Chapter 7, the more complex model detailed here adds specific hypothesised indirect effects.

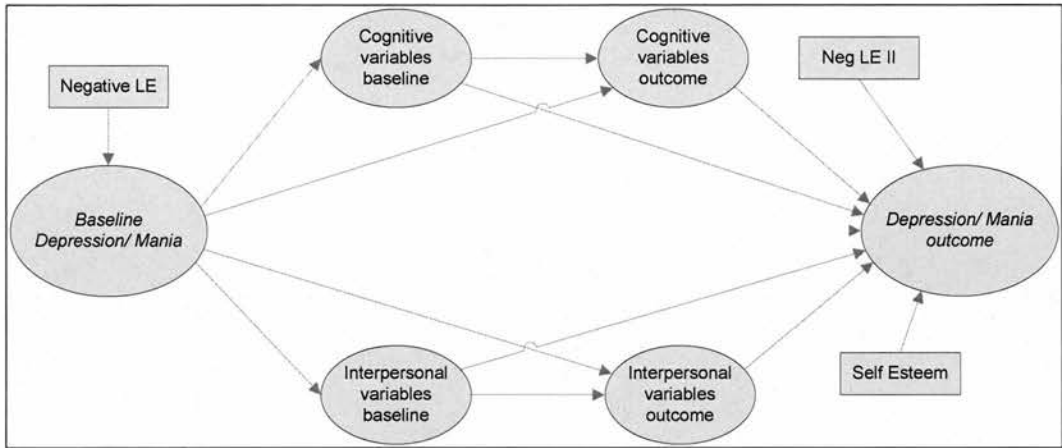


Figure 8.2: Schematic model of direct and mediation effects for key outcome variables

For the effect on outcome full mediational effect of cognitive and interpersonal factors are hypothesised with self esteem and negative life events prior to onset and negative life events prior to recurrence or residual symptomatology following treatment having an independent direct effect. Essentially it is hypothesised that the association between psychological and psychosocial variables at baseline and the same set of variables following psychological intervention fully mediate or ‘explain’ symptomatology after the intervention. As alternatives to the full mediational models mediation and direct effects are considered in the testing of the models.

8.2 Statistical Analysis

The primary analytic method chosen for these analyses was structural equation modelling (SEM) using EQS version 6.1 (Bentler, 2006). As described by Ullman (2006) SEM is a collection of statistical techniques that allow a set of relationships between one or more independent variables (IV), either continuous or discrete, and one or more dependent variables (DV), either continuous or discrete, to be examined. Both IV or DV can be

represented as factors or latent variables or measurement variables. Advantageously, SEM permits simultaneous assessment and prediction of several dependent variables within a single model. Specifically, SEM is a hypotheses testing or confirmatory approach to data analysis where a theoretical model of the relationship of dependent and predictor variables is hypothesised and subsequently tested how well the model 'fits' the data. Some variables used in the SEM analyses are not normally distributed. SEM is particularly robust against non-normal distributions and the robust model statistics are reported that are corrected for non-normal distributions. The correction used is the Satorra-Bentler robust correction (Bentler, 1996).

Goodness of fit of all models was evaluated using the Satorra-Bentler robust fit statistics: The Satorra-Bentler χ^2 (S-B χ^2) and the Robust Comparative Fit Index (RCFI: Bentler, 1998). The chi-square is the most commonly used measure of model fit and assesses the model's 'lack of fit'; a high chi-square value with a significant p value suggests a poor fit of the model to the data. The RCFI ranges from 0 to 1 with values greater than 0.90 indicating a good fit. The Root Mean Square of Approximation (RMSEA: Browne and Cudeck, 1993) is a measure of fit that takes into account a model's complexity where a RMSEA of 0.05 or less indicates a good model fit. Alternative fit indices commonly reported in SEM such as the Joreskog-Sorbrom's adjusted GFI are also reported.

Covariance SEM was utilised to examine the goodness of fit of four sets of a priori models relating general psychiatric symptoms, depression and mania as dependent variables to the cognitive, interpersonal and psychosocial variables. For all models the direct and mediating effects of the main hypothesised mediating factors were systematically tested. This test of mediation effects through SEM (Sobel, 1988) is more powerful than the mediating variable regression approach (Baron & Kenny, 1986;

Mackinnon et al., 2002). The only corrections to the models were based on hypothesised alternatives and the contribution of individual variables to the measurement constructs of the cognitive and interpersonal variables.

The specification of directionalities of presumed causal effects is a crucial part of SEM. The measurement of key variables over time provides one means to specify directionality and this is employed in the longitudinal SEM. When variables are concurrently measured at the same timepoint, then the specification of directionality in SEM requires a clear conceptual or theoretical rationale. Where the directionality of effects are conceptually uncertain, alternative path models are tested, each with alternating directionalities, in order to falsify specific assumptions about the directionality of effects. This method has to be balanced with the methodological implications model complexity and directionality should be conceptually specified a priori wherever possible. In the association between cognitive and interpersonal psychological variables in this study, alternative models of directionality have been tested in order to clarify empirically the conceptual arguments of interpersonal variables influencing the expression of core beliefs and assumptions and vice versa.

8.2.1 Methodological assumptions

In SEM a number of issues need to be considered. First, model identification: In SEM a model is specified and parameters for the model are estimated using the estimated population covariance matrix. But only models that are identified can be estimated. A model is said to be identified when there is a unique numerical solution for each of the parameters in the model. To ensure this the first step in the SEM analysis is to examine the measurement model underlying any structural SEM. The measurement model deals

with the relationship between the measured variables in the model and the confirmed maximum likelihood factors (CFA). Each factor should have at least two indicators, the more the better, and the error variances for each indicator should not be correlated. For this reason cognitive and interpersonal variables have been combined into fewer factors than measures employed in the outcome models as they related too closely to each other to justify separate factors.

After a model is specified the population parameters are estimated with the goal of minimising the differences between observed and estimated population covariance matrices. Following the specification and parameter estimation of the model the fit of the model is evaluated. As in regression analyses a good fit is sometimes indicated by a non-significant χ^2 . In SEM however, trivial differences between estimated and population covariance matrices are often significant; especially with small samples, the computed χ^2 may not be distributed as χ^2 , leading to inaccurate probability levels (Bentler, 1995) (for a detailed discussion see recent debate in volume 42 of *Personality and Individual Differences*, 2007). Because of this problem numerous measures of model fit have been proposed. The most commonly employed measures are the comparative fit index (CFI), estimating a range of models from the model corresponding to a set of completely unrelated variables to the perfect model with no degrees of freedom, the CFI places the estimated model on this continuum. This results in a fit index between 0 and 1 with values greater than 0.9 generally indicating a good fit of the model (Hu & Bentler, 1999). Another widely available fit index is the calculation of the weighted proportion of variance in the sample covariance accounted for by the estimated population covariance matrix. This goodness of fit index (GFI) is analogous to the R^2 in multiple regression and can be adjusted to the number of parameters in the model (AGFI). Finally there is the

index based on residuals, the root mean square residual expresses the average difference between sample variances and covariances and the estimated population variances and covariances. To aid interpretation the root mean square residual error of approximation (RMSEA) is provided; small values indicate a good fit of the model, values of .08 or less are desired (Hu & Bentler, 1999).

In addition to structural and measurement SEM it is also possible to test nested or multi group models. In this procedure of SEM separate identical models are run for each group in the sample (e.g. men and women or treatment modalities) and following the baseline model estimation models are then tested with parameters constraint across models to be equal. χ^2 statistics are then run between the two models to determine whether they are significantly different. This can be achieved separately for the measurement and structural part of the SEM, essentially determining whether the models between two groups differ in their basic factor structure or in their regression paths between the latent variables. Multi group SEM has been utilised in this analysis to determine whether the hypothesised outcome models differ between the two independent treatment modalities.

The sample for these analyses included all participants who entered treatment (N=193), missing values at the end of treatment were estimated using the EM procedure as implemented by EQS 6.1. The assumptions for each SEM model were evaluated using EQS. There were no univariate or multivariate outliers included and there was evidence that both univariate and multivariate normal distributions were violated for the baseline and outcome samples; Mardia's normalised coefficient was between 5.48 ($p < .005$) and 35.94 ($p < .001$) indicating violation of multivariate normality. Therefore the models were

estimated with maximum likelihood estimation and tested with the Satorra-Bentler scaled χ^2 .

8.3 Effects of predictors of bipolar symptoms at baseline

As outlined above in Figure 8.1 the direct and indirect effects of cognitive, interpersonal and psychosocial variables on bipolar symptomatology at baseline are evaluated. In the model several questions are of interest:

- 1) How well does the model explain the population covariance matrix?
- 2) How well to the constructs predict the measured indicator variables?
- 3) Do cognitive, interpersonal and psychosocial variables directly predict levels of bipolar symptomatology?
- 4) Are specific cognitive characteristics such as dysfunctional attitudes and personal beliefs about illness mediated by environmental and interpersonal variables?

8.3.1 Predictor model of general psychiatric symptomatology at baseline

For the factor of general psychiatric symptomatology no support was found for the hypothesised models of indirect effects; $\chi^2(325)=375.26$; $p < .001$; RCFI= .498; RMSEA= .181, or the alternate tested model of direct effects only (model of multiple regressions); $\chi^2(201)=88.15$; $p < .001$; RCFI= .688; RMSEA= .109.

Post hoc model modifications did not seem indicated given the extremely poor fit of the simple measurement model of multiple regressions, direct effects only. Also the

theoretical basis for the formulation of alternative specific hypotheses regarding the general psychiatric symptom level appeared very slim and further model development based on purely statistical indicators alone seemed contra indicated.

8.3.2 Predictor model of depression at baseline

For symptoms of depression the first hypothesised model tested was the model assuming full mediation of cognitive factors through current interpersonal variables and psychosocial indicators. This initial model had a poor fit: $\chi^2(309)=884.08$; $p<.001$; RCFI=.764; RMSEA=.083. As the measurement model alone showed a good representation of the population data and there were clear theoretical and statistical indicators as to how the model could be improved, post hoc modifications were performed and an alternative model was tested with a reduced number of indirect effects and clear direct effects of social support, negative life events and cognitive factors such as personal beliefs about illness (e.g. loss, shame and humiliation) and metacognitions (e.g. negative thoughts and uncontrollability), a model akin to multiple regressions with a simple mediation. This alternative model detailed in Figure 8.3 reached a moderate fit: $\chi^2(201)=684.08$; $p<.001$; RCFI=.881; RMSEA=.063.

Higher levels of symptoms of depression were predicted by negative life events (LEDS), social support (SOS) and metacognitions (MCQ). Higher levels of dysfunctional attitudes (DAS) and lower levels of self esteem did not predict higher levels of depression. This finding is surprising, although it illustrates the advantage of SEM over a series of independent multiple regression analyses. Both predictor models of depression, using social support and life events as one set of predictors and using dysfunctional attitudes and self esteem as another are significant. However, when considered together and in

interaction, the direct effects of self esteem and dysfunctional attitudes relinquish once a full measurement model is validated that links these psychological predictor variables through their association.

Further, there was a significant small mediation effect in that social support served as an intervening variable between levels of personal beliefs about illness (PBIQ; loss, humiliation and shame) and depression. Social support and personal beliefs about illness have a significant effect on the level of depression at baseline, but the product of the association between personal beliefs about illness and social support on depression outweighs the direct effect of personal beliefs on depression.

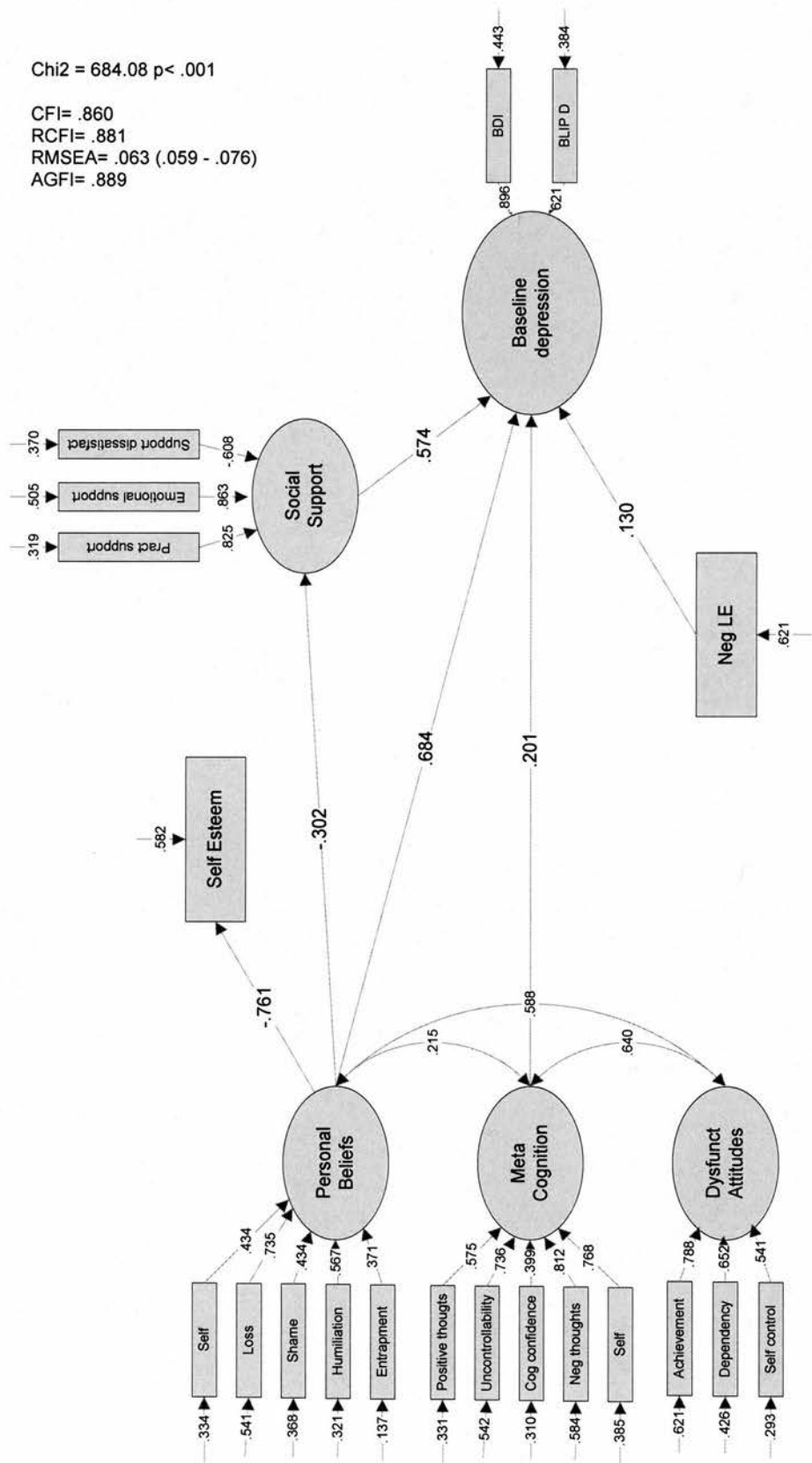


Figure 8.3: SEM of depression at baseline

8.3.3 Predictor model of mania at baseline

For mania at baseline changes had to be made to the initial measurement model as the latent factors for each of the cognitive and interpersonal measures could not be identified. Two new factors were therefore constructed comprising single factors for cognitive and interpersonal variables respectively. Further it was considered on theoretical grounds to include depression into the model due to hypotheses that the severity of manic symptoms may be dependent on underlying depression.

The first hypothesised model tested, assuming full mediation of cognitive and interpersonal variables plus a direct effect of negative life events only reached a moderate fit: $\chi^2(290)=406.08$; $p < .001$; $\text{RCFI} = .782$; $\text{RMSEA} = .071$. When depression however was added to the model a good fit of the model was reached: $\chi^2(204)=329.27$; $p < .001$; $\text{RCFI} = .899$; $\text{RMSEA} = .054$. In this model as illustrated in Figure 8.4 Higher levels of symptoms of mania were predicted by negative life events (LEDS), the cognitive factor (MCQ, PBIQ, DAS) and depression, with self esteem impacting on the cognitive factor. The model shows clear mediating effects of the impact of cognitive variables on mania being mediated by both recent negative life events and current depression, with the cognitive factor showing a main impact on concurrent depressive symptoms.

Chi2 = 329.27 p< .001

CFI= .886
RCFI= .899
RMSEA= .054 (.043 - .064)
AGFI= .906

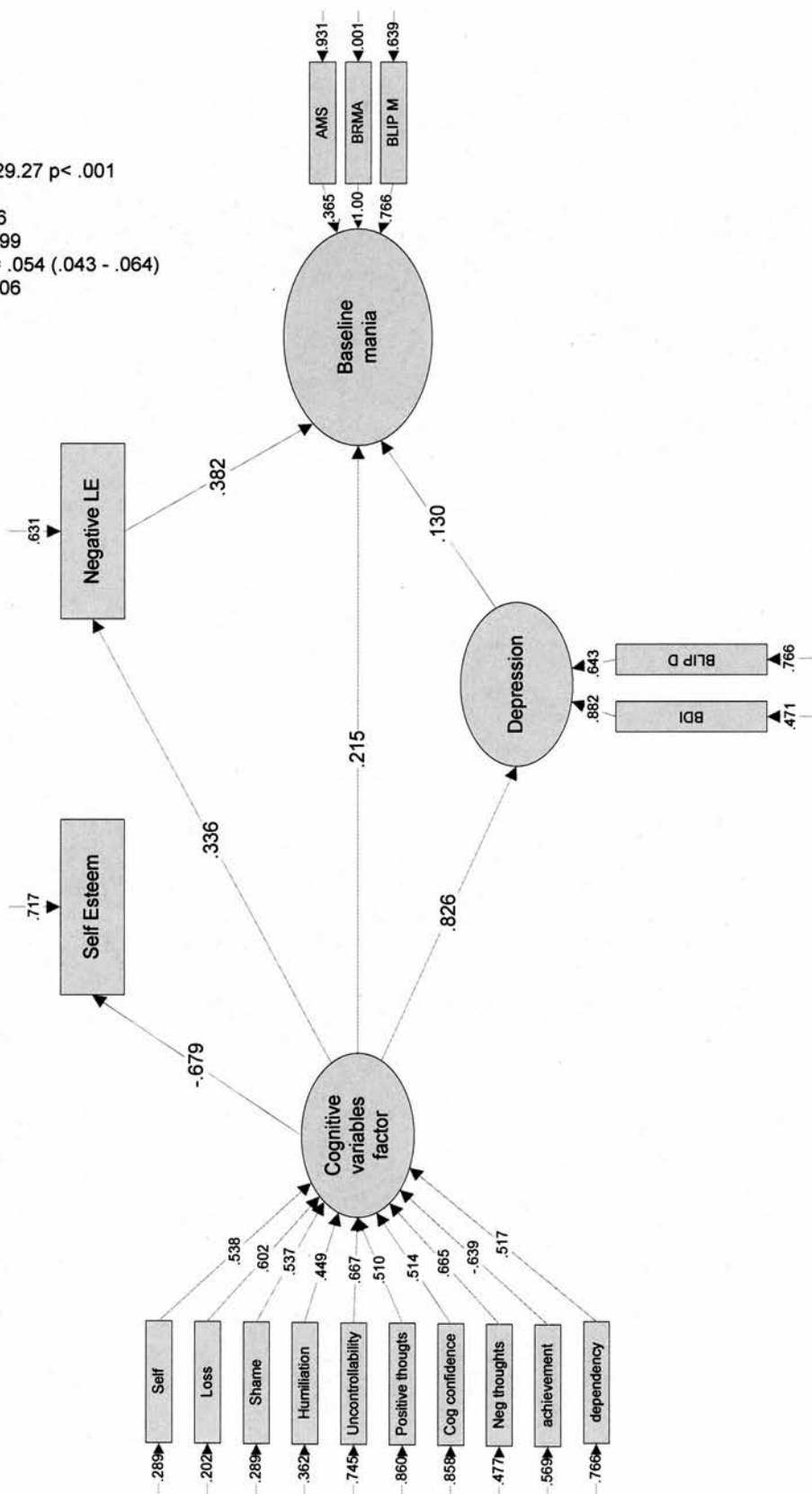


Figure 8.4: SEM of manic symptoms at baseline

8.4 Predictors of outcome

As illustrated in Figure 8.2 SEM was utilised in this step of the analysis to estimate the direct and indirect effects of baseline symptomatology as well as cognitive, interpersonal and psychosocial variables, including their associations, on outcome after psychological treatment. Specific questions asked were:

- 1) How well does the model explain the population covariance matrix?
- 2) How well to the constructs predict the measured indicator variables?

And, in particular:

- 3) Do cognitive, interpersonal and psychosocial variables at baseline and following treatment interact in their prediction of levels of symptomatic distress at outcome?
- 4) Are there specific mediating effects of cognitive and interpersonal variables?

8.4.1 Predictor model of general psychiatric symptomatology following treatment

As for the SEM model at baseline the hypothesised models for general psychiatric symptoms following treatment had an extremely poor fit: $\chi^2(552)=476.62$; $p < .001$; RCFI= .594; RMSEA= .089 and $\chi^2(704)=716.15$; $p < .001$; RCFI= .621; RMSEA= .079.

No further modification or interpretation of the models was indicated as they did not explain more for the general psychiatric symptoms outcome than the general multiple regression models described in Chapter 7.

8.4.2 Predictor model of depression following treatment

During the first step analyses, the measurement model underlying the longitudinal SEM in this section failed to differentiate cognitive and interpersonal constructs as defined by the individual measures, but instead the CFA verified four clearly distinct factors of cognitive and interpersonal variables combined at baseline and outcome.

The prediction of level of depression following treatment testing a mediational model of depression at baseline, and a combined cognitive factor and a combined interpersonal factor at baseline and following treatment taking into account self esteem and negative life events as independent predictors yielded interesting results. The initial model achieved a moderate fit: $\chi^2(416)=328.96$; $p < .001$; RCFI= .793; RMSEA= .055. This model however gave clear indicators as to the modifications necessary. Therefore the impact of negative life events was removed and interpersonal variables were specified as having a direct effect on depression. This generated a model that provided an excellent fit to the data: $\chi^2(394)=44.32$; $p = .053$; RCFI= .973; RMSEA= .024.

As detailed in Figure 8.5 the effect of baseline depression on depression outcome is clearly mediated by the factor of cognitive variables; with the combined effect of cognitive variables on depression and baseline depression on cognitive variables following treatment being significantly higher than that of the direct effect, with mediating effects explaining about 6 to 12 times the proportion of variance in contrast to the direct effect of depression at baseline on depression at outcome. Depression was further predicted directly by a negative interpersonal factor, dissatisfaction with current social support, perceived criticism and perceived emotional over involvement from close others. Self

esteem at baseline impacted directly on baseline depression and self esteem following treatment contributed directly to the cognitive factor, rather than depression outcome.

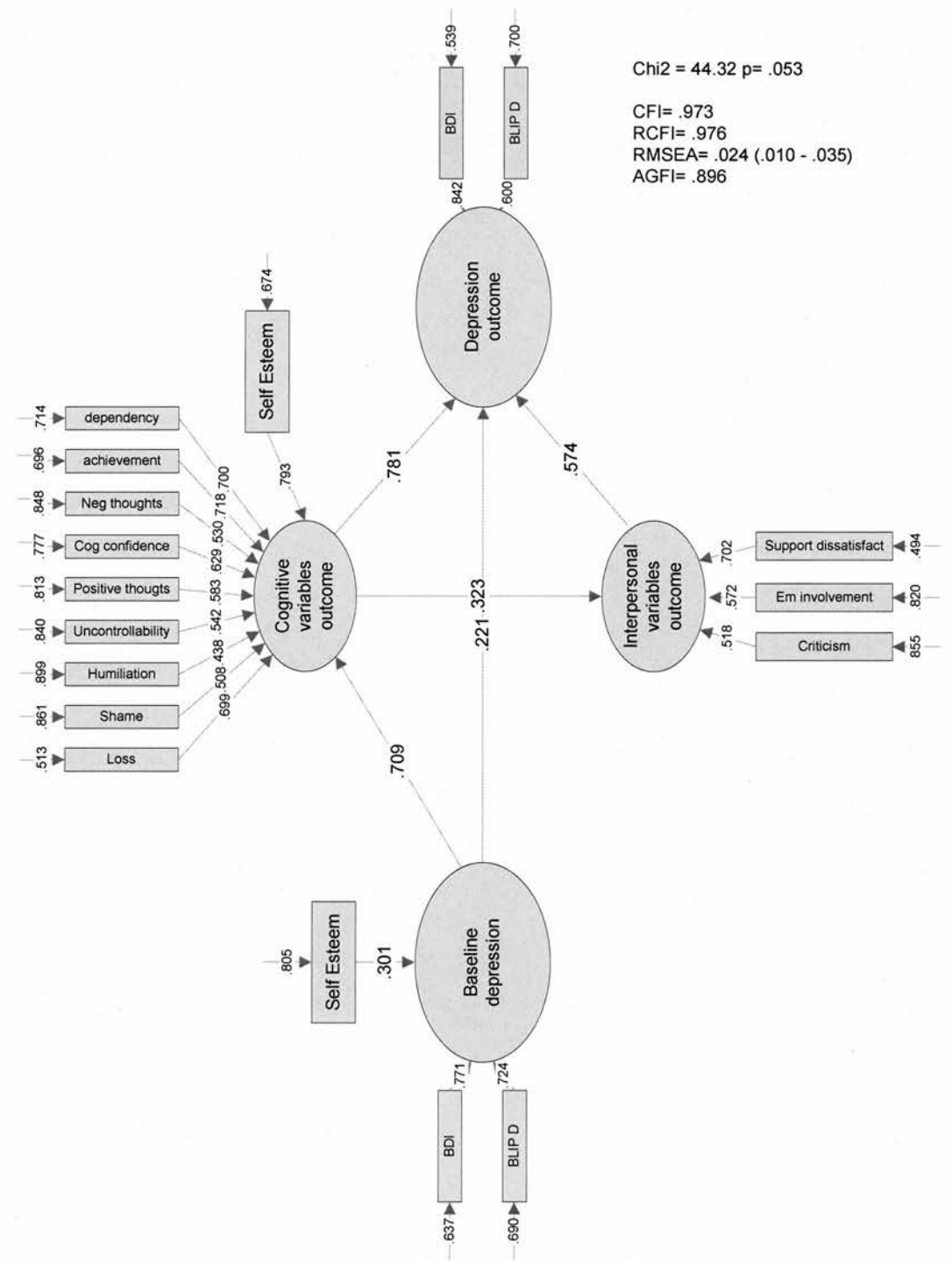


Figure 8.5: SEM of depression following treatment

8.4.3 Predictor model of mania following treatment

In line with the findings from the baseline predictor model depression at baseline was added to the prediction SEM for manic symptoms at outcome. The full mediational model including baseline characteristics for cognitive and interpersonal variables as well as direct effects for negative life events and self esteem resulted in poor model fit: $\chi^2(411)=492.18$; $p<.001$; RCFI= .774; RMSEA= .060.

Model modifications included a removal of baseline cognitive and interpersonal characteristics as well as mediating effects in a second step of modification in line with a simple regression model of multiple direct effects. The resulting model presented a very good fit to the data: $\chi^2(591)=177.92$; $p=.046$; RCFI= .918; RMSEA= .038.

As fully represented in Figure 8.6 the level of manic symptoms following the intervention effect was directly linked to cognitive characteristics following treatment as well as level of depression at baseline. There was also a mediating effect of the level of self esteem strengthening the effect of the cognitive factor on manic symptoms at outcome. Baseline symptomatology and interpersonal variables further contributed significantly to mania outcome but to a lesser degree.

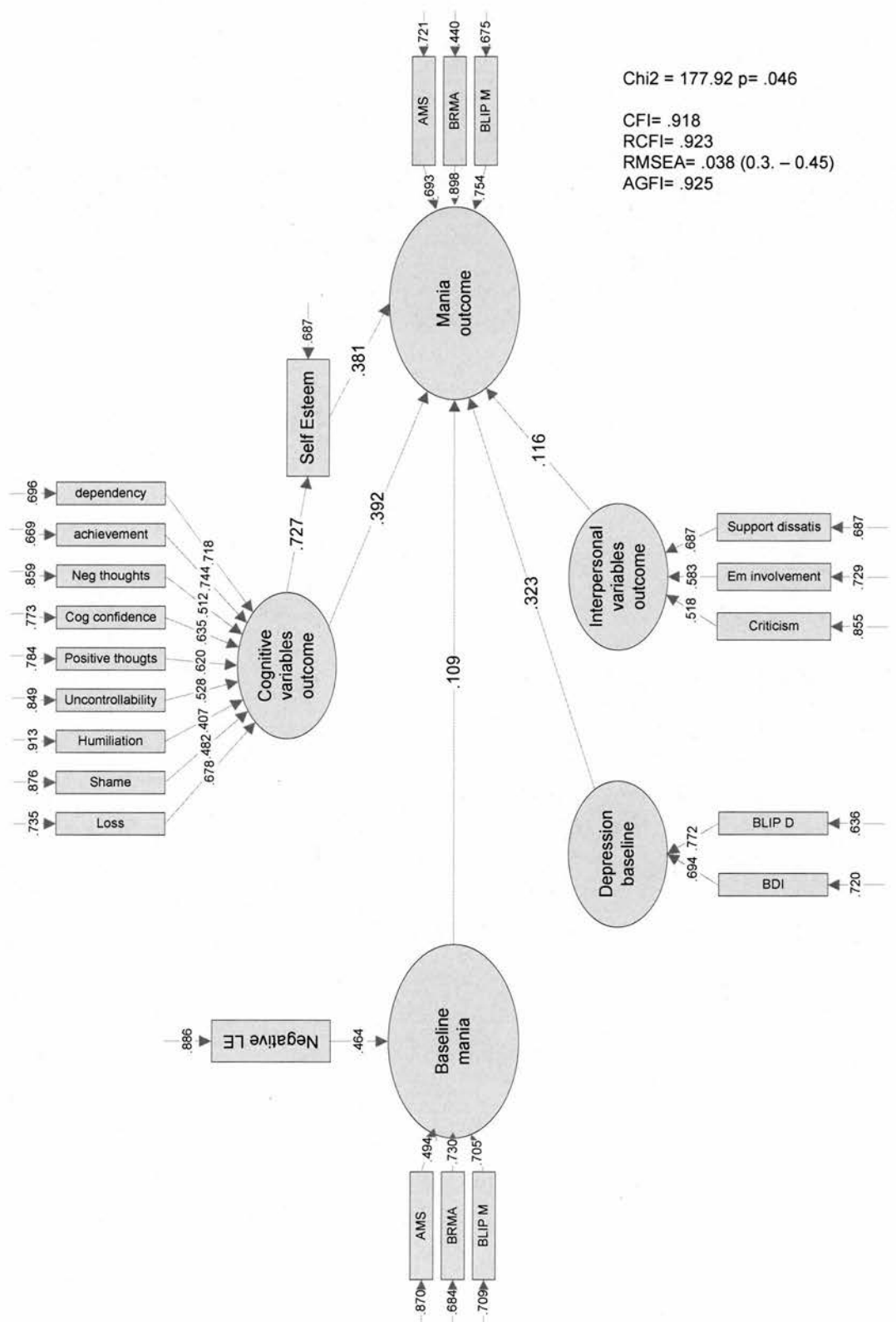


Figure 8.6: SEM of manic symptoms following treatment

8.5 Summary

The methodology of multiple regression analyses for the investigation of complex relationships between various dependent and independent variables is distinctly limited. Firstly it is only possible to investigate one dependent variable at a time which in a design with several related outcomes poses clear restrictions. Secondly even though complex relationships between independent variables in relation to the dependent variable such as singular mediation effects can be examined using a multiple regression framework, this necessitates multiple computations of regression analyses with an inflated number of covariates which inherently limits the power of such analysis. Also within such a methodology multiple mediating effects cannot be addressed and even though it will produce estimates for the paths between the variables investigated, the analysis will not indicate an overall fit of the model to the data and thus limiting its validity.

To avoid some of these shortfalls and to test the cohesiveness and power of the prediction models described in Chapter 7 a number of hypothesised structural equation models have been built and evaluated. The method of covariance modelling employed here further has the advantage that models of variable interactions are specified a priori and evaluated rather than explored based on empirical correlations. The modification of the structural models in the process of analysis was limited to theoretically based alternative hypotheses only. The measurement models were improved using statistical indicators of fit of individual variables.

Similar to the multiple regression analyses the structural equation models (SEM) were constructed in two steps: In a first step the associations and effects of interacting independent and dependent variables relating to the two affective dimensions of bipolar

disorder were tested, proposing interacting mediating effects between cognitive and interpersonal characteristics. In a second step fully mediational model of treatment effects were proposed, including complex relationships between dependent and independent variables at baseline and following treatment, investigating simultaneously whether effects of change in cognitive, interpersonal and psychosocial dimensions were predictive of outcome or alternatively these psychological characteristics at baseline or end of treatment.

In the analysis of the baseline models it became quickly apparent that models for general psychiatric symptoms could not be identified, which is consistent with the variable regressions models relating to this symptom dimension. The models for the manic and depressive symptom factors at baseline however show interesting results. For depression there was a clear mediating effect of social support for the cognitive predictors, indicating that the social environment and perceived support from others in availability and satisfaction clearly mediated the association between cognitive vulnerability factors and symptomatic distress. Further, the cognitive factors were highly intercorrelated and there was a strong indirect association between dysfunctional attitudes, metacognitive beliefs and beliefs about illness, and their overall prediction of depression once the interaction between these three factors could be considered. Also noteworthy that there is a further indirect contribution in the form of self esteem which appears to impact on self and illness appraisal strengthening its prediction of depression rather than with the depression dimension directly.

For mania it becomes apparent that the distinct cognitive dimensions are no longer preserved, but that the measurement model throws up that they are all related to one latent factor of appraisal and attribution and that in the relation to mania the

interpersonal variables are no longer predictive of level of symptoms. Also, in the structural model there are clear mediating and indirect effects; self esteem also indirectly impacts on levels of appraisals and attributions rather than on mania directly and there are significant mediation effects of negative life events and level of depression enhancing the relationship between cognitive variables and mania.

The apparent differences between these two structural models clearly indicate different and distinct pathways for manic and depressed symptoms in this sample of bipolar patients.

The directionality of the psychological predictors and mediators reported in the baseline and longitudinal SEM findings are in part set by the sequence of their measurements. Where associations and interacting effects between psychological constructs are considered and hypothesised in the specified models, these were either determined by a conceptual assumption, e.g. belief formation being influenced by interpersonal and developmental processes, or they have been tested empirically by specification of alternative models using changing and alternate directionality of effects.

For the outcome dimensions again only models for depressive and manic symptom dimensions could be identified and again the measurement models had to be reduced to individual latent factors for cognitive and interpersonal variables at baseline and at end of treatment, as the original measurement models preserving individual measures did not adequately fit the data. Also, originally hypothesised models of full mediation effects of cognitive and interpersonal variables at baseline and end of treatment did not provide a good enough fit of the data in the population.

For the depression dimension at the end of treatment there are clear mediation effects observable; baseline depression appears to be predictive of depression at outcome, but its association is significantly mediated by the cognitive dimension at end of treatment, following intervention and in turn the effect of these cognitive variables of appraisals and attributions on depression are mediated by the factor of negative interpersonal variables at outcome of expressed emotion and support satisfaction. The model that best explains the data for the depressed symptom dimension at the end of treatment is the model that hypothesised interacting mediation effects of cognitive appraisals and negative aspects of the interpersonal environment. In addition, self esteem appears to impact directly on levels of depression at baseline but only indirectly at outcome via its strong association with the cognitive variables.

Similar to the findings for the analyses at baseline a very different structural model converges for manic symptoms following treatment. It appears that levels of mania after the intervention are only mildly predicted by mania at baseline, but are mainly associated with levels of depression and to a lesser degree by the dimension of negative interpersonal characteristics. Cognitive variables of negative appraisals and attributions also contribute significantly to explain the level of manic symptoms after treatment but their effect is clearly mediated by the level of self-esteem. It appears, that with both levels of depression and low self esteem influencing levels of mania directly, negative affect as well as negative perception of the social environment have a major bearing on mania, rather than the hypothesised moderation of cognitive appraisals processes. Further it is noteworthy that in the measurement model for manic symptomatology positive and negative cognitive appraisals and beliefs did not form independent dimensions but converged on one factor, strengthening the argument that mania is influenced by negative and positive affect as well as negative and positive thinking styles

and beliefs in the face of stressors through negative life events and negative social environments.

Overall it appears that the confirmed structural equation models for baseline symptomatology and symptoms outcome enhance the level of interpretability of key predictors of bipolar vulnerability and treatment effects by their possibility to highlight and evaluate complex interacting effects.

IX Discussion and integration of findings

When this treatment trial was designed and implemented there was only a small number of clinical outcome studies in bipolar disorder focusing on the psychological treatment of bipolar symptomatology, medication adherence and coping strategies; the interventions described in these early studies were mainly psycho educational in nature and enhanced with behavioural strategies for dealing with the early development of symptoms and the behavioural consequences of manic and hypomanic mood states (e.g. Haas, *et al.*, 1988; van Geet & Zwart, 1991; and Honig *et al.*, 1997). There were also some early pilot studies utilising cognitive behavioural strategies, many of which were still focusing on medication adherence (Cochran, 1984; Hirschfeld-Becker *et al.*, 1998; and Perry *et al.*, 1999). During the first two years of the trial a couple of early studies were published that implemented a classic CBT model plus early warning signs monitoring (Lam *et al.*, 2000; and Scott *et al.*, 2001). In addition to these studies there were also some initial results from studies using Interpersonal and Social Rhythm Therapy (Frank *et al.*, 1999; Frank *et al.*, 2000) and Family Intervention specifically targeted at families of individuals with bipolar disorder (Miller *et al.*, 1991; and Miklowitz *et al.*, 2000). These early results seemed promising but often offered quite a narrow focus on relapse and recovery in bipolar disorder for example by concentrating on medication adherence or relapse prevention in euthymic bipolar populations.

The design of the treatment implemented in this trial was mainly inspired by the contemporary clinical developments in psychological interventions with individuals suffering from psychosis, and the developments in the psychological treatment of

recurrent and chronic depression. Rather than focusing on relapse and recurrence the treatment aimed at an improvement in the individual's understanding of their mood instability, their response styles and appraisals of negative and (hyper) positive affect, their mechanisms of affect regulation and their specific individual vulnerability factors in terms of past negative and traumatic life events, the impact of the disorder on their personal and psychological development, and their social and interpersonal aspects of vulnerability to mood instability and negative affect regulation.

In addition to the primary aim to evaluate an integrated and formulation based treatment combining cognitive behavioural therapy elements and interpersonal psychotherapy components in terms of its efficacy and effectiveness in a randomised clinical trial, the study further aimed to explore key psychological and psychosocial variables to aid the development of a model driven clinical understanding of the vulnerability, onset and recovery in bipolar disorders.

9.1. Trial design and implementation

From the start, the study was set up in close collaboration with service users and carers through the involvement and support of the Bipolar Fellowship Scotland (BFS), and latterly the Manic Depression Fellowship. Members of the BFS were involved in discussions of the trial design and treatment format. Members of the BFS also assisted in piloting the assessments and supported the development of the psychological intervention by sharing their experiences of psychotherapeutic support and psychosocial interventions. The service users in the group were very familiar with a psychoeducation

concept that was used throughout Scotland in the development of a peer support group network over the last 10 years.

9.1.1 Patient preference

The trial employed a mixed or nested design of randomisation to psychological treatment (CIT+TAU) plus treatment as usual, and treatment as usual alone (TAU). Once randomised to psychological treatment participants had a choice between individual psychological intervention and a group format. Individual treatments consisted of 16 to 20 weekly one to one sessions that lasted about one hour and groups consisted of 20 weekly sessions of 90 minutes length. At the outset our expectation was that the majority of participants in the trial would prefer individual treatment and that we would possibly run only two groups per year.

The literature on patient choice specifically for group or individual treatments is mixed. Prouty et al. (2002) found that young women with eating disorders when offered a choice would prefer individual support as their first choice and group treatment as their second choice, this was in part related to symptom severity in that individuals with more severe symptoms preferred group format as their first choice over individual support. Alvarez and Azocar (1999) asked patients of a gynaecology clinic who showed high levels of emotional distress about their interest in psychotherapy and a majority of those surveyed showed interest in individual psychotherapy over group psychotherapy or group psychoeducation. Dwight-Johnson et al. (2000) however found in a sample of depressed primary care patients that 47% of their sample preferred group counselling over individual counselling when offered a choice.

Contrary to our expectations we found that from the start of the trial about half of participants opted for group treatment, which necessitated us running two groups per week in parallel during the first year of the study. One reason for the large proportion of participants opting for group treatments may have been linked with the promotion of peer support groups throughout Scotland by the BFS at the time. We know that many of the individuals who participated in the trial had prior experience of groups within user and carer organisations including the BFS. We were aware that at the time both the BFS and other user organisations were lobbying for improved access to psychological treatments for users of psychiatric services. Other factors that may have biased patient choice in this sample towards a more equal distribution of interest in group versus individual treatment are experiences of inpatient and day patient care of most of our participants. In both of these settings group formats are frequently used for different aspects of psychosocial interventions. Another reason may be the wish for mental health service users to reduce stigma and to find commonality in their distress (McCay et al., 2007), something that is readily facilitated and experienced within group treatment settings.

The aspect of the trial design that included patient preference for the modality in which they would receive treatment has a number of dimensions relating to aspects of service delivery and how users can engage in mental health services. This aspect of the trial may also improve treatment reliability especially in relation to psychotherapeutic group treatments. However, there are also methodological implications to consider in terms of a partially randomised trial design.

In terms of treatment integrity, we felt at the start of the trial that randomly allocating participants to group or individual treatment would not allow the facilitation of an

effective interactive group format where individuals need to bring a willingness to work with people and to acknowledge openly some of the difficulties that led them to seek psychological treatment. Apart from the ethical dimension of an enforced treatment modality we also thought that in providing a psychological therapy service for individuals suffering from bipolar disorder we should allow people to choose between the available treatment options as a model of service delivery and to support retention in the group treatments. It is well documented that service users' choice of available treatments improves retention in mental health settings (e.g. Dwight-Johnson et al., 2000; Dalal et al., 2007). This was strongly supported by service users and carers on the project steering group who felt that individuals should have access to their preferred treatment modality and that consequently they would engage more effectively with the intervention. The steering groups also felt very strongly that the element of patient choice within a research trial does reflect the reality of many clinical settings. This is particularly true of those who follow best practice recommendations and guidelines, where service users may be offered a choice of treatments between which they would express an informed preference, and this decision would be based on sufficient information regarding the different available treatment options.

However, it was important to consider the methodological implications of introducing patient preference into the trial design. Essentially, the option of participants to choose between the two treatment modalities after allocation to treatment or after the waiting list control condition, meant that the trial was only partially randomised to treatment and control group, but not fully randomised in terms of psychological treatments offered. Randomisation procedures are performed in order to maximize the internal validity of treatment outcome studies. Objections have been made that this practice undermines the external validity of these studies because it ignores patients' treatment preferences,

thereby precluding the self-selection of treatment that can occur in the community (Bower et al., 2005; King et al., 2005). A number of studies were carried out to investigate the impact of randomisation compared to patient choice or patient preference on a range of outcomes. Leykin et al. (2007) reviewed findings of a randomised controlled trial for depression using two groups, medication and cognitive behavioural therapy, where participants were randomised to either treatment after also stating a treatment preference. They found that participants expressed preference compared to randomisation had no effect on the reduction of depressive symptoms; either condition was comparably effective in reducing depressive symptoms, although participants allocated to treatment that was not their stated preference were more likely to discontinue treatment. King et al (2005) carried out a systematic review of the impact of participant intervention preference in RCT's in general. They concluded that although treatment preferences led to a substantial proportion of people refusing randomisation or discontinuing treatments, differences in outcome across the trials between randomised and preference groups were generally small, particularly in large trials and after accounting for baseline measures of outcome. Therefore, there was little evidence that preferences substantially interfere with the internal validity of randomised trials.

9.1.2 Retention and involvement of participants

As reported in Chapter 5 we experienced a very high retention rate over the treatment phase with only 14% of participants dropping out before the minimum number of session, 67% being available for the first follow-up assessments after 6 months and 54% being available for the final follow up at 18 months. This finding is in line with comparable intervention trials where groups experienced attrition rates of 12% (Morrison et al., 2004), 14% (Rosenberg et al., 2004), 17% (Turkington et al., 2002) or 20% (Miklowitz et

al., 2003) in targeted psychological or psychosocial intervention trials and up to 27% (Miklowitz et al., 2006) in control groups. Across these studies lower attrition rates appear to be associated with trials where psychological interventions are delivered independently and where these are not delivered in the context of psychiatric services.

There are a number of factors that are likely to have supported the high retention of participants in this study. We felt that patient choice in the treatment plus randomisation to the delayed treatment condition rather than an alternative 'placebo' treatment aided retention as individuals felt that they received the treatment that they had requested. Another important factor was the user and carer organisation involvement in and support of the study. Many individuals who participated in the trial learned about the project from BFS meetings and local peer support groups. In addition the patient council of the Royal Edinburgh Hospital had an initiative at the time lobbying for better access to psychological treatments for people suffering from severe and enduring mental health problems in Lothian. In that respect it was very obvious to participants referred to the project that this was a service developed in line with users' requests and their involvement, and the start of the project seemed very timely. This is structure of service delivery that is comparable to the targeted psychological intervention service offered in Manchester to individuals at ultra high risk of developing psychosis where attrition was at 12% in the treatment group (Morrison et al., 2004).

In addition, because all treatment was offered from within the project team and therapists were employed for this purpose, we were able to offer extremely flexible clinics and assessments. Most of the groups were run in the early evenings in group facilities in the Royal Edinburgh Hospital or the local mental health community base. Individual sessions were scheduled to suit the participants other commitments and many

clinics were offered out of hours. Further, most of the follow up assessments took place in community bases or in the context of home visits. Finally, we learned from the follow up interviews with participants that one of the most positive aspects of the project was the level of additional support offered, in that many individuals claimed that routinely they were not seen very frequently by their key workers or psychiatrists and that these contacts were often focused on symptoms and their medication treatment regime.

9.2 Key outcomes of cognitive interpersonal therapy in bipolar disorder

In Chapter 6 the main treatment effects were examined. In sum, the direct comparison with the waiting list control group showed a large treatment effect of psychological intervention on the primary outcome, quality of life. Similarly, medium to large treatment effects were established for the secondary outcomes, on all indicators of bipolar symptoms, emotional distress and indicators of relapse and recurrence. In the systematic comparison over time individuals who received a course of the integrated cognitive interpersonal intervention either in an individual or group therapy treatment format showed an evident reduction in bipolar symptoms and a significant increase in their quality of life. On the whole these primary treatment effects were maintained at six months and eighteen months follow up. There was some indication however that on measures of subsyndromal affective distress the treatment effects were not fully maintained at the eighteen months follow up. On indicators of hospital admissions and use of psychiatric services individuals who completed treatment showed a significant reduction in service utilisation, inpatient admissions and length of inpatient stays compared to their pattern of service use before and after treatment and compared to the control group within the six months control interval.

The integration of clinical efficacy and effectiveness is still difficult in the field of clinical bipolar disorder research as most trials to date focus on different indicators of outcome. Lam and his group (Lam *et al.*, 2003; and Lam *et al.*, 2005) used time in episode and time to recurrence as indicators of effectiveness in their trial of CBT for euthymic bipolar individuals. Scott and colleagues (Scott *et al.*, 2006) evaluated outcome in a large trial of a mixed bipolar population by medication adherence and recurrence rates of bipolar episodes. Ball *et al.* (2006) examined mania and depression scores following CBT treatment and medication adherence. Frank and colleagues (Frank *et al.*, 2000; Frank *et al.*, 2005) implementing IPSRT in a combination trial with cross randomisation of IPSRT, psychoeducation and clinical management including long term maintenance support looked at recovery time from manic or depressed episodes and survival times until recurrence.

The heterogeneity of indicators is partly due to the developmental status of the field and a lack of consensus what objectives and aspirations psychological interventions may feasibly have in bipolar disorders; reduced relapse rates, symptom reduction, longer survival times to recurrence, or constructive maintenance, 'damage limitation' and productive coping with mood instability.

Overall the outcomes of the clinical research trials and pilot studies thus far are mixed. Most of the earlier trials of psychological interventions in small pilot samples and relatively homogeneous clinical groups achieved positive results in terms of survival rates to recurrence, length of time without bipolar symptoms. Lam and colleagues (2005) reported in a recent 30 months follow up of their original sample of 103 remitted bipolar patients lasting effects of cognitive therapy intervention on mood ratings, social

functioning and coping with early and milder bipolar symptoms. However, similar to the IPSRT trial by Frank and colleagues (2005), Lam et al. found that the effects are largely attributable to the lengthened time to recurrence of depressive episodes.

The largest randomised controlled multi centre trial by Scott et al. (2006) including 235 patients with bipolar disorder in 5 centres and Ball et al.'s (2006) recent CBT trial of 52 mixed bipolar I and bipolar II patients however achieved no lasting results in their main outcome variables, recurrence rates and lower symptom ratings. Scott and colleagues did not find a significant reduction in recurrence in the treatment group when compared to the control group. The research group found that this negative finding was mainly attributable to patient characteristics and consequently the heterogeneity of their sample which in contrast with many of the previous trials included patients with current substance misuse, high frequency of recurrence and high levels of co-morbidity. They also conceded that the type of intervention used follow a classical CBT model primarily developed for non recurring episodes of major depression and that issues in relation to treatment fidelity monitoring across centres and therapists may have impeded the individual focus of the treatment for each of these complex cases. A similar observation can be made about Ball's trial where treatment followed a relatively traditional format with a clear focus on psychoeducation, behavioural strategies, early signs monitoring and modification of dysfunctional beliefs. Ball and colleagues achieved positive treatment effects but found that those could not be maintained over the 12 month follow up period, arguing that for non-remitted bipolar patients' some form of ongoing psychological maintenance treatment is indicated.

In light of the recently reported evidence it may seem surprising that this study found very robust and sustained positive treatment effects from psychological intervention in a

mixed group of bipolar patients with relatively high symptom ratings at the start of treatment compared to other trial samples. Some of the advantageous features of the psychological treatment in this study may be the integrative nature of the interventions combining cognitive therapy elements, with interpersonal psychotherapy, and a strong therapeutic focus on established vulnerability factors in bipolar disorder, such as a focus on specific dysfunctional beliefs (Alloy *et al.*, 2005; Tzemou & Birchwood, 2006), traumatic past events (Garno *et al.*, 2005) and associated intrusive emotional resonances, as well as interpersonal and social influences in terms of social supports (Cohen *et al.*, 2004) and expressed emotions (Miklowitz *et al.*, 2005).

There may further be an argument that psychological intervention for complex disorders such as bipolar disorder, psychosis or personality disorders need to remain highly individualised and governed by comprehensive psychological formulation of individuals' needs and goals, and therapists' responsiveness to therapeutic processes, in a way that makes their implementation in large multi-centre studies very difficult where considerations of treatment fidelity and minimisation of therapists' effect are often dominant.

9.3 Psychological models of bipolar disorder

As reviewed in Chapter 3, a consistent range of psychological factors and processes has been examined in the context of bipolar disorder. The main concepts contributing to psychological theories of bipolar disorder are circadian rhythm instability, the role of depression in the genesis and recurrence of mania (manic defence), the role of specific dysfunctional beliefs and attributional styles to mood instability and mood fluctuation, the

role of significant negative life events and trauma, and the influence of social and interpersonal environments and processes such as social support and expressed emotion. Often these contributing processes are pursued in isolation in order to specify certain effects and crystallise a particular model or test a distinct theoretical assumption.

In this study the attempt has been made to evaluate key psychological variables relevant to the vulnerabilities and impairments in bipolar disorder within an integrative model where their direct and indirect effects could be examined. The three main conceptual perspectives included were: the role of social support and negative life events, the significance of dysfunctional beliefs and cognitive styles and attributions, as well as the influence of affective states and the individual's attempts at affect self regulation.

9.3.1 Predictors of bipolar symptoms

The main predictors of symptomatic distress are distinctly different for the two symptom dimensions. In the current study the two dimensions of affective symptoms, levels of depression and mania, are associated with negative life events and key cognitive factors relating to cognitive confidence, self esteem and shame for depressive symptoms and cognitive confidence and negative metacognitive beliefs for manic symptoms.

In the analyses relating to the prediction of the key outcome indicators there is also a distinctively different pattern of variables impacting on the dimensions of bipolar symptoms at the end of treatment compared with the significant psychological and psychosocial predictors at baseline. Significant negative life events no longer have a strong association with levels of bipolar symptoms and there are more significant interpersonal variables as well as cognitive characteristics. Depression now is strongly

associated with loss, humiliation and shame as well as symptoms at baseline and metacognitive beliefs about controllability. Manic symptoms on the other hand are predicted by positive metacognitive beliefs, cognitive confidence and self control as well as the criticism dimension of expressed emotion.

In respect of dysfunctional beliefs and negative attributional styles Johnson & Fingerhut (2004) found in a prospective study of 60 individuals with bipolar I disorder that dysfunctional attitudes and negative automatic thoughts were significantly associated with depression but not mania. Other authors, for example Bentall and Thompson (1990) suggested that mania may be triggered by negative affect and associated negative cognitions, and Reilly-Harrington and colleagues (1999) found that negative cognitions were a predictor of hypomania in a sample of bipolar spectrum participants. Johnson and Fingerhut are therefore suggesting that hypomania and mania in bipolar disorders results not only from negative affect and negative cognitive styles but from an interaction between negative cognitions and events related to threat, which would help to explain the interaction between negative life events and negative cognitions for mania in this sample. A parallel argument can be made in relation to the increase in cognitive confidence being predictive of mania in bipolar individuals (Johnson, 2005) and its potential interaction with threatening and negative life events.

In line with the literature on cognitive factors in bipolar disorder the findings of the present study confirm the relevance and importance of dysfunctional attitudes (Lam et al., 2004; Johnson & Fingerhut, 2004), particularly in relation to goal attainment beliefs and dependency. The findings also demonstrate the need for specific cognitive concepts and measures as the constructs of dysfunctional attitudes, illness related personal beliefs and metacognitions were developed within particular disorder groups, namely depression,

schizophrenia and anxiety disorders, and without a full transdiagnostic validation. The measurement models in Chapter 8 suggest that the validity and specificity of these construct could not be confirmed for this population. Since the designs and implementation of the trial there have been considerable advances in the development and validation of bipolar specific cognitive scales. Mansell (2006) and Mansell and Jones (2006) developed the Hypomanic Attitudes and Positive Predictions Inventory (HAPPI), to identify the specific cognitions relating to hypomania and mania. Jones et al. (2006) developed the Hypomania Interpretations Questionnaire (HIQ) which consists of two subscales, positive self-dispositional appraisals and normalising appraisals. Both measures successfully differentiate bipolar from control groups in these cognitive characteristics and aim to be able to assess and target particular cognitive styles in bipolar patients to further enhance cognitive therapy interventions for this group. It is interesting to note that on an item level both measures contain a number of other appraisals, specifically items relating to personal autonomy and perceived understanding and empathy in others; this may be further confirmation of a strong interpersonal element in the mechanisms of mood dysregulation in bipolar disorder as highlighted in the findings of the present study.

It is interesting to note that despite the strong theoretical and empirical support for the robust influence of social support and negative interpersonal styles, or expressed emotion, the variables relating to these concepts did not feature in the linear hierarchical regression analyses once cognitive variables have been taken into account. This could either suggest that the recorded influence of social and interpersonal variables are a proxy for appraisal processes in bipolar disorder when these are not assessed at the same time, or that there are strong interactive effects that are not accounted for with the present methodology. This is reflected in findings of attributions linked to expressed emotions in relatives of schizophrenic patients, where social environments marked by

highly critical or emotionally overinvolved others are associated with specific causal and internal attributions (Barrowclough & Hooley, 2003). This indicates that processes of appraisals and attributions are mediated by interpersonal factors and that the quality of key relationships in the social network influences the formation of appraisals. Further, Lam et al. (2004) found that goal attainment attitudes were higher in individuals with bipolar disorder than in individuals with unipolar depression, and that people with bipolar disorder also reported higher levels of 'anti-dependency' beliefs. This was linked to interpersonal styles of individuals with bipolar disorder in these samples. Lam et al. (1999) found that bipolar patients often seek out others for stimulation and validation of their ideas and beliefs, particularly in periods of ascent to mania, and that they also find it much harder to accept advice from others in elevated mood states (Mansell & Lam, 2006). These findings indicate that interpersonal factors are much more closely related to key cognitive indicators than is suggested in their linear relationship and suggests that interpersonal factors may well act as mediating factors of the association of key cognitive variables, such as dysfunctional attitudes and beliefs about illness, bipolar symptoms and outcome.

9.3.2 Integration of cognitive and interpersonal factors in bipolar disorder

In order to examine possible interacting and mediating effects Chapter 8 described the testing of structural and measurement models of the main cognitive interpersonal and psychosocial variables described.

At baseline in the cross sectional models of prediction of bipolar symptoms there were clearly differentiating findings between the models for depression and mania. In the depression model there was a clear mediating effect of social support for the cognitive

predictors, indicating that the social environment and perceived support from others in availability and satisfaction mediated the association between cognitive vulnerability factors and symptomatic distress. Further, the cognitive factors were highly intercorrelated and there was a strong indirect association between dysfunctional attitudes, metacognitive beliefs and beliefs about illness, and their overall prediction of depression once the interaction between these three factors could be considered. There also was a further indirect contribution in the form of self esteem which appears to impact on self and illness appraisal strengthening its prediction of depression rather than on depression directly.

For manic symptoms on the other hand it became apparent that there were clear mediating and indirect effects in the structural model; self esteem also indirectly impacted on levels of appraisals and attributions rather than on manic symptoms directly and there were significant mediation effects of negative life events and level of depression enhancing the relationship between cognitive variables and mania.

These findings of the structural models for levels of depression and mania respectively provide a strong basis for the argument of different pathways and mechanisms for the expression and recurrence of manic and depressive symptoms in bipolar disorder.

In relation to the specific influence of social support in bipolar disorder this is consistent with existing findings. Johnson and colleagues (1999) suggested a polarity specific effect in that social support influences the expression of depression but not mania in a prospective study of 59 individuals with bipolar I disorder. They similarly found analogous to the findings in the present study that there was no significant interaction effect of life events and social support in bipolar participants, despite the mounting evidence of this

interaction effect in unipolar depression (Champion, 1995). It is interesting however to note that in the present study life events are a mediator for the influence of cognitive appraisals on manic symptoms which may account for some of the differences that have been noted in the interaction between negative life events and psychosocial factors (Johnson, 2005).

Similarly for the association between self esteem and depression; Johnson and colleagues (2000) found in a prospective study of 31 bipolar one participants that self esteem and social support were not linked to the expression of manic symptoms, but predicted depression independently of social support in an analysis of the hypothesised interaction effect between the two variables. In this study it was also found that self esteem was independent of social support in relation to the prediction of depression, but once cognitive factors have been included there is only an indirect effect on depressive symptoms via negative cognitions, beliefs and appraisals.

In respect of the prediction of symptoms of depression following treatment there are clear mediating effects: Baseline depression appears predictive of depression at outcome, but its association is mediated by the levels of negative beliefs and appraisals at end of treatment, and the effect of these cognitive variables on depression are mediated by negative interpersonal variables in the form of expressed emotion and support satisfaction. The model that best explains the data for the depressed symptom dimension at the end of treatment is the model that hypothesised interacting mediation effects of cognitive appraisals and negative aspects of the interpersonal environment. In addition, self esteem appears to impact directly on levels of depression at baseline but only indirectly at outcome via its strong association with the cognitive variables.

For mania, levels of manic symptoms after the intervention are only insubstantially predicted by levels of mania at baseline, but are mainly associated with levels of depression and to a lesser degree by the dimension of negative interpersonal characteristics. Cognitive variables of negative appraisals and attributions also contribute significantly to explain the level of manic symptoms after treatment but their effect is clearly mediated by the level of self-esteem. It appears, that with both levels of depression and low self esteem influencing levels of mania directly, negative affect as well as negative perception of the social environment have a major bearing on mania, rather than the hypothesised moderation by cognitive appraisals processes. Further, for manic symptoms positive and negative cognitive appraisals and beliefs converged on one factor, strengthening the argument that mania is influenced by negative and positive affect as well as negative and positive thinking styles and beliefs in the face of stressors through negative life events and negative social environments.

These models of manic and depressive symptoms following treatment confirm the findings that negative interpersonal styles are predictive of manic and depressive outcomes in bipolar disorder (Miklowitz *et al.*, 2005), but that these are complex interactions that follow different psychological mechanisms for mania and depression respectively. This is in line with recent findings by Thomas *et al.* (2007) in a study of manic patients, depressed bipolar patients, remitted bipolar patients and healthy controls, that found that response styles to depression is the key differentiating mechanism in this group with manic patients showing greater use of active coping and risk taking which is likely to be associated with positive metacognitive beliefs and self esteem.

One of the most surprising finding is the mediating effects of social support on levels of depression, both in relation to the baseline model and the model of depression

outcomes. In line with the strong literature on the effects of social support and life events on depression, reviewed in Section 2.6, a clear direct effect of social support on depression, or alternatively a clear mediation effect of social support on the association between negative life events and depression, may have been predicted. Equally, the effects of cognitive factors and those of social factors could be expected to be found in correlated, but separate, pathways in relation to depression or mania. Johnson et al. (2000) found that social support appeared to be the most important predictor of change in depression but not mania, and Johnson and Fingerhut (2004) concluded equally that cognitive measures, such as the Dysfunctional Attitudes Scale and the Automatic Thoughts Questionnaire, predicted increase in depression over time but not mania. In isolation these studies seem to suggest separate psychological mechanisms for mania and depression in bipolar disorder and separate pathways for cognitive and social support related variables. What makes these findings difficult to interpret is that there have been extremely few studies that combine interpersonal and cognitive variables in the study of the psychology of bipolar disorder.

The current findings, however, seem to suggest that there is a strong interaction between key attributional styles, appraisals and beliefs related to depression in individuals with bipolar disorder and that the effect of cognitive factors is mediated by social support variables. Possible explanations include that key cognitive factors, such as key attributional styles, appraisals and beliefs are neither formed nor activated in isolation and in the context of purely internal psychological processes. Both developmentally and contextually, key interpersonal experiences play a major role in the development of individuals' key beliefs and attributional styles and that therefore the current interpersonal context would have an essential influence on how cognitive factors may impact on mood and how depressogenic cognitive processes may be balanced or

corrected by close others (Johnson, 2005; Tzemou & Birchwood, 2006). Another possible interpretation is that the mechanisms of affect regulation in bipolar disorder can be understood as a dynamic interaction between internal and external factors. Gottschalk et al. (1995) pointed out that mood variation in bipolar disorder is not cyclical, nor completely random, but results from a complex interaction between internal vulnerability variables and independent external variables over time. This finding has been picked up by a number of authors more recently in the context of the development of psychological models of bipolar mood variation within which increasingly sensitive and biased information processing develops through successive dynamic interaction with the environment over time and through an accumulation of negative illness related experiences (Mansell et al. 2007; Jones, 2001). It appears in this connection that the study of interpersonal styles and mechanisms of affect regulation over time in relation to bipolar disorder could benefit from more systematic research.

9.4 Theoretical implications

As discussed in Sections 2.6 to 2.8, the theoretical understanding of the psychological mechanisms involved in bipolar disorder is restricted. Key models relate to increased sensitivity in individuals with bipolar disorder to neuropsychological behavioural activation systems in relation to goal attainment life events (e.g. Johnson et al. 2005; Wright and Lam, 2004) and to the destabilising impact of circadian rhythm disruptions (Jones, 2001). Separate from these neuropsychological models of mood regulation, authors focused on cognitive styles and cognitive biases in bipolar disorder that influence compensatory behavioural strategies which are thought to contribute to the

development of mania (e.g. Bentall, 2003; Leahy, 2005). These models however remain very unintegrated and are predominantly tested independent of alternative assumptions.

Mansell et al. (2007) recently put forward an integrative conceptual model of mood swings in bipolar disorder that aims to put together the empirical evidence for alternative models of affect in bipolar disorder. They suggest a functional pattern where the interpretation of intrusions and internal mood states leads to paradigmatic response styles in bipolar disorder that are driven and catalysed by dysfunction in the behavioural activation system, a vulnerability to circadian rhythm disruptions and specific cognitive appraisal processes in the face of perceived mood changes. These self-reinforcing patterns can then quickly spiral into the development of mania and depression through specific 'ascent behaviours' (e.g. risk-taking, increased alcohol and drug use, etc.) or 'descent behaviours' (e.g. social withdrawal, rumination, etc.) which are triggered in the individual as an attempt to control unwanted mood states. The authors emphasise that this describes a model of mood regulation that also applies to similar difficulties in related disorder groups, such as psychosis and schizoaffective disorder, and that is not specific to bipolar disorder. A key aspect of this model appears to be the misinterpretation of internal states (physiological, emotional or cognitive) as bearing significant personal meaning and with a number of contradictory appraisals. These appraisals of changes in internal states can include them being signs of an imminent catastrophe, a personal success or a personal weakness. These appraisals then trigger immediate efforts at exerting control. The model further proposes that the appraisal of changes in internal states and the associated ascent/ descent behaviour are influenced by a range of factors, such as personal core beliefs regarding self and others and beliefs about affective states. These beliefs are affected by past experiences and current stressors.

The empirical findings of the present study highlight some of these proposed interacting processes, especially the role of cognitive appraisals mediating the effects of social and stress related factors differentially for manic and depressive symptoms in bipolar individuals. The findings of the present study further suggest that personal appraisals that influence individual reactions to changes in internal states are not only influenced by past experiences and current stressors in general, but specifically by the quality of past and current relationships that influence core beliefs related to early mechanisms of affect regulation, such as the development of interpersonal trust and the establishment of stable confiding relationships and the internal representation of others as basically reliable and trustworthy. These patterns of interpersonal styles and their developmental correlates of attachment and reflective function require further systematic research in order to establish their specific role in affect regulation in bipolar disorder.

The main clinical implications of this perspective are the active integration of interpersonal styles and behaviours within cognitive behavioural formulation and treatment strategies in bipolar disorder, specifically the linking of the processing of interpersonal experiences and difficulties and current difficulties in regulating negative affect and unwanted mood changes. For details on the particular clinical framework and techniques see Gumley and Schwannauer (2006).

9.5 Limitations of the present study

In the considerations of the findings of the present study a number of limitations ought to be considered. Despite our efforts to keep all research assistants who carried out baseline and follow-up assessments blind of treatment condition and the time point in the

schedule, i.e. whether participants presented before the TAU assessment or when they were reassessed when starting treatments, complete blindness could not be maintained throughout the study. Further, participants were randomised into psychological treatment plus psychiatric management or psychiatric management alone; no true control treatment condition over the whole course of the study was provided. The results of the treatment trial should therefore be seen as exploratory requiring replication in a fully randomised and fully blinded controlled clinical trial. In a recent review of the effectiveness of 24 cognitive behaviour therapy trials for schizophrenia Wykes et al. (2007) noted that trials without full randomisation and masking of assessments can be overoptimistic about the effects of cognitive behavioural therapy and overestimate main treatment effects with masked studies demonstrating an estimated effect size of 0.30 and unmasked studies of 0.49.

In terms of the ongoing psychiatric management the project team had no influence over changes in medication management, we requested the RMO of each participant to aim to keep medication levels and treatments stable and the same over the course of treatment but we know from the case note analysis that for a significant subgroup medications were changed during the participation in the trial. There was little evidence however from this data that the medication management was systematically influenced by individual's participation in the psychological treatment trial.

Also, all psychological treatments were carried out by two therapists and the results may reflect therapist effects. Adherence to the treatment manual was enhanced by weekly supervision; however, no formal assessments of treatment fidelity were carried out. At the time when the study and the intervention was designed and implemented there was no evaluated treatment manual for the psychological treatment of bipolar disorder

available and especially during the pilot phase of the study the treatment format developed considerably with the feedback from participants. The first three individual patients and the first group were therefore viewed as experimental and data from these participants are not included in the analysis.

9.7 Future research

The trial highlighted a number of aspects in the psychology of bipolar disorder that merit further research. In terms of conceptual questions it raised important questions regarding our understanding and measurement of essential risk factors in bipolar disorders, particularly in terms of the integration of different research traditions concerned with social and psychosocial risk in life events and social networks on the one hand and cognitive risk factors on the other. Particularly the interaction between the development and formation of personal beliefs and appraisals and key interpersonal processes requires further attention in order to specify how developmentally key social interactions and attachment experiences shape both cognitive processing of external and internal stimuli and how these feed into the fundamental capacity for affect regulation. In this respect we have started a number of collaborative research studies examining the influence of attachment and reflective function in individuals with a first onset of psychosis and bipolar disorder and how these developmental factors relate to coping strategies and cognitive factors.

A second area for future research is the clinical application of psychological interventions in this clinical group. The trial has highlighted that a highly targeted and individualised psychological intervention combining cognitive therapy and interpersonal therapy can be

very beneficial for individuals with a bipolar disorder. This raises important questions of the application and delivery of psychological interventions for this client group and how transferable the findings of the trial are into a NHS setting. Since the completion of the project we had the opportunity to carry out a number of training events for Community Mental Health Teams and Clinical Psychologists in the NHS and we aim to develop a case series to further refine the psychological interventions and strategies used and to investigate further which aspects appear to be particularly helpful for individual service users.

X Papers originating from the present study

Cavanagh, J., Schwannauer, M., Power, M. & Goodwin, G.M. (submitted). A novel scale for measuring mixed states in bipolar disorder. *American Journal of Psychiatry*.

Gumley, A.I. & Schwannauer, M. (2006). *Staying Well After Psychosis: A Cognitive Interpersonal Approach to Recovery and relapse prevention*. Chichester: John Wiley & Sons.

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Appendix

Semi-structured Interview Guide and Assessment Tools

Interview Schedule

INTERVIEW SCHEDULE

Date: ____/____/____

Initials: _____

No.: _____

First I want to ask about a few basic details and about the people you live with and those you have contact with.

Can I begin by asking your date of birth? ____/____/____

Age:

Gender:

Are you married?

— living with somebody?

Date of start of current marriage/ cohabitation: ____/____/____

Have you ever been married before or lived with anyone for more than six months?

No. of marriages:

No. of cohabs. (> 6 months):

Who else lives with you at present?

No. of household members including S:

Do you have any children?

No. of children in household:

No. of children born to subject:

Are all the children yours and your husband's/ partner's?

If in question - ?

Excluding the people that live with you, how many relatives live within 20 miles?

With whom are you in regular contact?

Excluding the people that live with you, how many friends live within 20 miles?

With whom are you in regular contact?

Are you employed at present?

if unemployed: **What year did you last work?**

Is it your choice not to work outside the home?

if employed: **Can you tell me what you do? — Do you have any official title?**

How long have you been in this job?

How many jobs did you have since you left school?

Is (spouse or partner) working?

Title:

Determine who is the main breadwinner.

How long have you lived in your current home?

Note housing type

Who owns the property you live in?

After you left school have you ever done a full time/ part time educational or training course?

☐ No ☐ Yes; What?:

What year did you begin your first course:

What year did you finish your last course (completed):

If no, - **Did you do any exams before you left school?**

Do you have any general health problems that stop you from doing things you must do?

If yes please say what kind of problem:

Do you drink any alcohol?

How much per week?

Do you use any recreational drugs or any other substances?

If yes, what do you take?

How often do you take them?

Did you experience any early childhood losses?

What?

When?

Since when have you experienced difficulties with your mental health, e.g. feel low and depressed, etc.?

At what age did you have your first serious mood swing?

At what age were you first diagnosed as having manic depression?

How many episodes of depression have you had?

When did your last episode of depression begin?

How many episodes of mania have you had?

When did your last episode of mania begin?

How long did it last?

To what extent did this episode disrupt your daily life?

Did your mental health get in the way of what you felt able to do?

more recently?

Have you ever been admitted to hospital for either mania or depression?

If yes,

- a. How many times in the last 5 years?
- b. How long on each occasion?

What treatments have you been given in hospital?

Have you ever been prescribed lithium?

What other medications have you been prescribed?

Has your medication been changed/ reviewed in the past 12 months?

How often do you see your doctor, your psychiatrist/ psychologist?

Are there other professionals you see regularly (nurses, social worker, etc.)?

Would you like to see more (semi)-professionals (befriender, volunteer support worker etc.)?

What other non-medication treatments have you received?
(e.g. group or individual therapy)

What treatment(s) do you perceive as being most effective for you?

Are there any forms of treatment you have not received but would like to?

How would you describe your relationship with your carer?

In your opinion to what extent does your carer understand:

- a. You?
- b. Your illness?

Do you know any members of your family, who have had difficulties with their mental health, e.g. were depressed?
who/ what:

Have they been in hospital for that reason, - or seen a specialist (Psychiatrist, Psychologist)?

If you would like to be considered for the follow up study we also need the following details:

If you choose not to fill in these details: We would be grateful if you could nevertheless complete the questionnaire. You have the option of sending it back anonymously.

Name:
Address:

Home Tel. No:

Relative contact/carers in own household.

Name:
Relationship:

Combined PANSS & BPRS assessment interview

About the *Hybrid Interview Schedule*

The "***Hybrid Interview Schedule***" is an integrated clinical symptom interview which allows the user to obtain all the necessary information to rate several commonly used scales. It integrates the Brief Psychiatric Rating Scale (BPRS), the Positive and Negative Syndrome Scale (PANSS). By eliminating the overlap between different scales, the Hybrid Interview Schedule provides a comprehensive assessment of the various symptom dimensions found in individuals presenting with psychosis. The frequency, severity, interference with functioning and other relevant aspects of the symptom are assessed over the preceding 4-week period. The information gathered during the interview is to be noted on the respective "***Hybrid Interview Schedule Recording Sheet***". The individual manuals for the above mentioned scales are to be used to rate each scale.

INTRODUCTION

"We are meeting together for about half an hour to an hour to get a better idea of the nature of the problems and symptoms that you may have had over the past month. I will be taking notes from time to time. This is simply so that I will be able to remember things later.

First of all, I wonder if you could tell me about how you have been doing in the past month. I'd like you to take a few minutes to tell me about your current living circumstances and your present well-being."

CONFIDENTIALITY (for non-PEPP patients only):" Everything you will say during our meeting will essentially remain confidential. Nevertheless certain things may come up that we believe should best be shared with your case manager/psychiatrist. Depending on the nature of such information I am also legally bound to share that information with your case manager/psychiatrist."

1. SOMATIC CONCERN

Establish :

- **FREQUENCY**
- **IMPAIRMENT OF FUNCTIONING**
- **DELUSIONAL**
- **DISCLOSURE**

- Have you been concerned about your physical health?
- Have you had any illness or seen a medical doctor lately?
- Has anything changed regarding your physical appearance? Is there anything wrong with your body?
 - Have you told anyone about this concern?
 - How often are you concerned about [X]?
 - Has it interfered with your ability to perform your usual activities?

Score BPRS 1, PANSS G1

- How have you been sleeping? Have you had any trouble falling asleep at the beginning of the night? Have you felt at any times like you needed less sleep than usual? If yes, do you feel rested when you wake up?
- Do you wake earlier in the morning than is normal for you?

Score HAS 11, YMRS 4, CDS 7

2. †ANXIETY

Establish :

- **FREQUENCY**
- **IMPAIRMENT OF FUNCTIONING**
- **AUTONOMIC ACCOMPANIMENT**

- Have you been worried a lot in the last [2wks] (What do you worry about?)
- Are you concerned about anything? How about finances or the future?
- How much of the time have you been [X]?
- Has it interfered with your ability to perform your usual activities/work?
- When you are feeling nervous, do your palms sweat or does your heart beat fast? (Shortness of breath/trembling/choking?)

Score BPRS 2, PANSS G2

Extra Anxiety Questions – needed to rate the Hamilton Anxiety Rating Scale

- Have you been feeling tense? Do you startle easy? Cry easily? Easily fatigued? Have you been trembling or feeling restless or unable to relax?
- Have you been feeling fearful (phobic) of situations or events? For example, have you been afraid of the dark? Of strangers? Of being left alone? Of animals? Of being caught in traffic? Of crowds? Other fears?
- Have you been experiencing aches, pains or stiffness in your muscles? Have you experienced muscle twitching or sudden muscle jerks?
- Have you been experiencing ringing in your ears, blurred vision, hot or cold flashes, feelings of weakness or prickling sensations?
- Have you had episodes of a racing, skipping or pounding heart? How about pain in your chest or fainting feelings?
- Have you been having trouble with your breathing? For example, pressure or constriction in your chest, choking feelings, sighing or feeling like you can't catch your breath?
- Have you had any difficulties with stomach pain or discomfort? Nausea or vomiting? Burning or rumbling in your stomach? Heartburn? Loose bowels? Constipation? Sinking feeling in your stomach?
- Have you been experiencing urinary difficulties? For example, have you had to urinate more frequently than usual? FOR WOMEN: Have your periods been regular?
- Have you been experiencing flushing in your face? Getting pale? Lightheadedness? Have you been having tension headaches? Have you felt the hair rise on your arms, the back of your neck or head, as though something had frightened you?

3. †DEPRESSION/Hopelessness/Self Depreciation

Establish :

- **FREQUENCY**
- **SEVERITY**
- **IMPAIRMENT OF FUNCTIONING**

- How has your mood been recently/in the last [X]?
- Have you felt depressed, sad or down?
- How would you rate it out of 10, with 10 the best you've ever felt, 0 the worst and five average?

- How often have you felt like that?
- When you have felt depressed have you noticed the depression being worse at any particular time of day?
- Are you able to switch your attention to more pleasant topics when you want to?
- Do you find that you have lost interest in or get less pleasure from things that you used to enjoy?
 - How long do these feelings last?
 - Has it interfered with your ability to perform activities/work?
- How do you see the future for yourself? Have you given up or does there still seem to be some reason for trying?
- What is your opinion of yourself compared to other people? Do you feel better or not as good or about the same as most?

Score BPRS 3, PANSS G6, CDS 1, 2, 3, 6

4. SUICIDALITY

Establish :

- **FREQUENCY**
- **IDEATION**
- **PLANS/ATTEMPTS**

- Have you felt that life wasn't worth living?
- Have you thought about harming yourself or killing yourself?
- Have you ever felt tired of living or as if you'd be better off dead?
 - How often have you thought about it?
 - Did (do) you have a specific plan?

5. GUILT

Establish :

- **PREOCCUPATION**
- **IMPAIRMENT OF FUNCTIONING**
- **DELUSIONAL**

- Is there anything you feel guilty about?
- Have you been thinking about past problems?
- Do you tend to blame yourself for things that have happened?
- Have you done anything you're ashamed of?
- Have you ever felt that you have done some terrible thing that you deserve to be punished for?
 - How often have you been thinking about [X]?
 - Have you disclosed your feelings of guilt to others?
- Do you have the feeling that you are being blamed for something or even wrongly accused?

Score BPRS 5, PANSS G3, SAPS 10, CDS 4 & 5

6. HOSTILITY

Establish :

- **ACTUAL BEHAVIOUR**
- **External & Internal HOSTILITY**

- How have you been getting along with people lately?
- Have you been irritable or grumpy? (Do you keep it to yourself? Do you show it?)
 - Were you ever so irritable that you would shout at people or start fights?
 - Have you hit anyone recently?
 - Have you done anything to try to harm animals or people?

Score BPRS 6, PANSS G14 , YMRS 5 & 9

7. ANHEDONIA-ASOCIALITY

Establish :

- **ACTIVE AVOIDANCE vs PASSIVE WITHDRAWAL**
- **INTEREST, DESIRE & INITIATIVE** (regarding activities, relationships)
- **ABILITY TO DERIVE PLEASURE/ ENJOYMENT** from activities, relationships

Impersistence at work/school:

- What is your primary activity? Do you work/attend school/homemaker?
- Do you enjoy your work/school, (*other chores*)?
- During the past month, have you been having any problems at work/school etc?

Physical Anergia/Hyperactivity:

- Are there times when you lie or sit around most of the day?
- What about the opposite, of finding that you have much more energy than normal, ie feel hyperactive or restless?
- Does this ever last longer than a day?

Occupational/ Recreational interests and activities/:

- What kinds of things do you do in your free time? Do you have any particular hobbies you enjoy like sports, political or religious activities, cooking, reading or any favorite TV programs?
- During the last month, what types of recreational activities have you engaged in?
- How often do you do these activities?
- Have you felt interested in the things you usually enjoy? (Have they been as fun as usual)?
- During the last month, have you had a chance to socialize with your friends or family? How often?
- Do you generally make the plans to go out or do you wait for somebody else to invite you?

9. GRANDIOSITY

Establish :

- **SEVERITY**
- **CONVICTION (acted on?)**
- **DELUSIONAL**

- Is there anything special about you? Do you have any special abilities or powers?
- Do you feel you are going to achieve great things?
- Have you thought that you might be somebody rich or famous?
 - How often have you been thinking about [X]?
 - Have you told anyone about what you have been thinking?
 - Have you acted on any of these ideas?

Score BPRS 8, PANSS P5, SAPS 11

10. SUSPICIOUSNESS

Establish :

- **FREQUENCY**
- **PREOCCUPATION**
- **DELUSIONAL**

- Do you ever feel uncomfortable in public? Does it seem as though others are watching you? Are you concerned about anyone's intentions toward you?
- Do you feel in any danger?
- Have you felt that people are against you? Is anyone going out of their way to give you a hard time trying to hurt you?
- Do you think people have been plotting against you?
 - How often have you been concerned about [X]?
 - Have you told anyone about these experiences?

Score BPRS 9, PANSS P6, SAPS 8 Score PANSS G16 (combine information from P6, G14, N4)

11. †HALLUCINATIONS

Establish :

- **FREQUENCY**
- **IMPAIRMENT OF FUNCTIONING**

Auditory hallucinations:

- Have you heard any sounds or people talking to you when there was no one around?
- What does/do the VOICE(S) say? Does it have a voice quality? Male/female?
- Do you ever hear your name being called?

Voices commenting:

Relations with Friends and Peers

- Do you have many friends? Close friends? How often have you seen them in the past month?
- Do you spend most of your time alone or with other people?
- Do you prefer to be alone?
- Do you join in activities with others? Do you enjoy being with others?
 - Why not? Are you afraid of people, or do you dislike them?

Ability to feel intimacy and closeness:

- Do you feel close to your partner or anyone in your family? Is there anyone that you feel particularly close to?
- Are you close to your family? How often do you see/hear from them? Do you keep in contact with them or do you wait for them to contact you?
- Do you have someone you can talk to if you need help? Do others confide in you?

Sexual activity and interest:

- Are you currently married/single?
- Do you currently have an intimate relationship/partner/boyfriend or girlfriend?
 - IF YES: How often have you seen him/her in the past month?
Did you enjoy your time together?
Have you noticed a decrease or increase in your interest in or enjoyment of sex?
 - IF NO: Would you like to meet someone?
Would you like to have a relationship/partner/ boyfriend or girlfriend?
 - IF NO: Why not? Do you have trouble meeting people?
 - IF YES: During the last month, were you interested in having sexual relations?
Have you noticed a decrease in your interest in or desire for sex?

Score PANSS N2, N4, SANS 15, 18, 19, 20, 21, YMRS 2 & 3

8. ELEVATED MOOD

Establish :

- **FREQUENCY**
- **SEVERITY**

- Have you felt so good or high recently that other people thought you were not your normal self?
- Have you been feeling cheerful or "on top of the world" without any reason?
 - Did it feel like more than just feeling good?
 - How long did it last?

Score BPRS 7, YMRS 1

- Have you ever heard voices commenting on what you are thinking or doing? What do they say?

Voices conversing:

- Have you heard two or more voices talking with each other? What did they say?

Visual hallucinations:

- Do you ever have VISIONS or see things that others don't see? What did you see?
- Did this occur when you were falling asleep or waking up?

Somatic or tactile hallucinations:

- Have you ever had burning SENSATIONS or other strange feelings in your body?

Olfactory hallucinations:

- Have you ever experienced any unusual SMELLS or ODORS that others don't smell? What were they?

For each hallucination reported, ask the following questions:

- Have these experiences interfered with your ability to perform your usual activities/work?
- How do you explain them?
- How often does this happen?

Score BPRS 10, PANSS P3, SAPS 1-6

12. †DELUSIONS

Establish :

- **FREQUENCY**
- **IMPAIRMENT OF FUNCTIONING**
- **CONVICTION/EXPLANATION**

Delusions of Jealousy

- Have you ever worried that your husband/wife might be unfaithful to you?
 - What evidence do you have?

Religious Delusions

- Are you a religious person?
- Do you have a special relationship with God?
- Have you had any unusual religious experiences?
- What was your religious training as a child?

Delusions of Reference

- Have you ever walked into a room and thought people were talking about you or laughing at you?
- Have you seen things in magazines or on TV that seem to refer to you or contain special messages for you?
- Are anything like X-rays, or radio waves affecting you?

Delusions of Being Controlled

- Have you ever felt that you were being controlled by some outside force?
- Have you felt that you were under the control of another person or force?

Delusions of Mind Reading

- Have you ever had the feeling that people could read your mind?
- Can anyone read your mind?

Thought Broadcasting

- Have you ever heard your own thoughts out loud, as if they were a voice outside your head?
- Have you ever felt your thoughts were broadcast so other people could hear them?

Thought Insertion

- Have you ever felt that thoughts were being put into your head by some outside force?
- Are thoughts put into your head that are not your own?

Thought Withdrawal

- Have you ever felt your thoughts were taken away by some outside force?

For each non-/delusional belief reported, ask the following questions:

- How often do you think about [X]?
- Have you told anyone about these experiences?
- How do you explain the things that have been happening?

Score BPRS 11, PANSS P1, G9, SAPS 9, 12, 13, 14, 15, 16, 17, 18, 19, 20

13. BIZARRE BEHAVIOUR

Social and Sexual Behaviour:

- Have you done anything that seemed unusual or disturbing to others?
- Have you done anything that could have got you in trouble with the police?

Clothing and Appearance

- Have you noticed anything unusual about your appearance?
- Have you done anything that has attracted the attention of others?

Repetitive or stereotyped behaviour

- Are there any things that you do over and over?

Score BPRS 13, SAPS 21-24

14. SELF-NEGLECT

- How has your grooming been lately? How often do you change your clothes?
- How often do you bathe/shower?
- Has anyone complained about your grooming or dress?
- Do you eat regular meals?

Score BPRS 13, SANS 14

15. †DISORIENTATION

- May I ask you some standard questions we ask everybody?
- What is today's date?
- What is this place/building?
- Who is the current Prime Minister?

Score BPRS 14, PANSS G10

16. INATTENTIVENESS

- "Can you spell the word "world" backwards?"(count errors)
- "Starting at the number 100, please subtract backwards by 7s (for those with at least Grade 100 or by 3s (for those with at least Grade 6) for a series of 5 subtractions." (count errors)

Score SANS 24

17. ABSTRACT THINKING

APPENDIX A : Items for assessing SIMILARITIES

<ol style="list-style-type: none"> How are a ball and an orange alike? Apple and banana? Pencil and pen? Nickel and dime Table and chair? Tiger and elephant? Hat and shirt? Bus and train? Arm and leg? Rose and tulip? Uncle and cousin? The sun and the moon? Painting and poem? Hilltop and valley? Air and water? Peace and prosperity? 	<p style="writing-mode: vertical-rl; transform: rotate(180deg);">Circle the Similarities used.</p>	<p>Note on Appendix A : Similarities are generally assessed by <u>sampling four of the items at different levels of difficulty</u> (i.e., one item selected from each quarter of the full set). When using the PANSS longitudinally, items should be systematically alternated with successive interviews so as to provide different selections from the various levels of difficulty and thus minimize repetition.</p> <p>Notes on Similarities responses :</p>
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You've probably heard the expression, "Carrying a chip on the shoulder." What does that really mean? There's a very old saying, "Don't judge a book by its cover." What is the deeper meaning of this proverb? »

APPENDIX B Items for assessing PROVERB INTERPRETATION

<p>What does the saying mean:</p> <ol style="list-style-type: none"> "Plain as the nose on your face" "Carrying a chip on your shoulder" "Two heads are better than one" "Too many cooks spoil the broth" "Don't judge a book by its cover" "One man's food is another man's poison" "All that glitters is not gold" "Don't cross the bridge until you come to it" "What's good for the goose is good for the gander" "The grass always looks greener on the other side" Don't keep all your eggs in one basket" "One swallow does not make a summer" "A stitch in time saves nine" "A rolling stone gathers no moss" "The acorn never falls far from the tree" "People who live in glass houses should not throw stones at others" 	<p style="writing-mode: vertical-rl; transform: rotate(180deg);">Circle the Proverbs used.</p>	<p>Note on Appendix B : Proverb interpretation is generally assessed by <u>sampling four of the items at different levels of difficulty</u> (i.e., one item selected from each quarter of the full set). When using the PANSS longitudinally, items should be systematically alternated with successive interviews so as to provide different selections from the various levels of difficulty and thus minimize repetition.</p> <p>Notes on Proverb responses :</p>
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18. LACK OF JUDGEMENT AND INSIGHT

- How long have you been in the hospital/clinic etc?
- Why did you come to the hospital/clinic etc?
- Did you need to be in a hospital/clinic etc?
 - i. Did you have a problem that needed treatment?
 - ii. Would you say that you had a psychiatric or mental problem?
 - iii. Can you tell me about it and what it consists of?
- In your opinion, do you need to be taking medicine?
 - i. *if medicated*: Why then are you taking medication?
 - ii. *if unmedicated*: Why are you still in the hospital/clinic etc?
 - iii. Does this medicine help you in any way?
- Do you at this time have any psychiatric or mental problems?
 - i. For what reason are you still in the hospital/clinic etc? Please explain.
 - ii. Just how serious are these problems?
- *if hospitalized*: Are you ready for discharge from the hospital?
 - i. Do you think you'll be taking medication for your problems after discharge?
 - ii. What are your future plans?
 - iii. What about your longer-range goals?

Score PANSS G12

"Well, that's about all I have to ask of you now. Are there any questions that you might like to ask me? Thank you for your co-operation."

END

Legend:

† indicates items for which ratings on the BPRS and PANSS are equivalent. BPRS and PANSS items are rated using a 7-point scale.

Interview Schedule Recording Sheet

Recording sheet for Patient's direct VERBAL RESPONSES to Questions

NB: Where necessary for rating (indicated by *), note down REPORTS

Subject No : _____ Date: _____ Interviewer: _____

INTRODUCTION	1. SOMATIC CONCERN
2. ANXIETY	3. DEPRESSION
4. SUICIDALITY	5. GUILT
6. HOSTILITY	7. *ANHEDONIA-ASOCIALITY

8. *ELEVATED MOOD	9. GRANDIOSITY
10. SUSPICIOUSNESS	11. HALLUCINATIONS
	Auditory/Visual/Somatic/Olfactory
12. DELUSIONS	13. *BIZARRE BEHAVIOUR
Jealousy/Religious/Reference/Control/Mind Reading/Broadcasting/Insertion/Withdrawal	
14. *SELF-NEGLECT	15. DISORIENTATION

16. INATTENTIVENESS	17. ABSTRACT THINKING
“WORLD”, Serial 3s or 7s	Similarities, Proverbs
18. LACK OF INSIGHT	COMMENTS:

Items based on OBSERVATION during interview and REPORTS from primary care workers, family or chart

1. FORMAL THOUGHT DISORDER	2. BLUNTED AFFECT
Derailment	Unchanging facial expression
Tangentiality	Decreased spontaneous movements
Incoherence	Paucity of expressive gestures
Illogicality	Poor eye contact
Circumstantiality	Affective non-responsitivity
Pressure	Inappropriate Affect
Distractibility	Lack of vocal inflection
Clanging	
Other observations: (e.g. neologisms):	
3. ALOGIA	4. *MOTOR BEHAVIOUR
Poverty of Speech	Physical Anergia
Poverty of Content of Speech	Motor retardation

<p>Stereotyped thinking</p> <p>Blocking</p> <p>Increased Latency of Response</p>	<p>Tension</p> <p>Excitement</p> <p>Motor Hyperactivity</p> <p>Poor Impulse Control</p> <p>Mannerisms and Posturing</p> <p>Disturbance of Volition</p>
<p>5. *RAPPORT/ENGAGEMENT</p>	<p>COMMENTS:</p>
<p>Preoccupation</p> <p>Uncooperativeness (interpersonal compliance)</p> <p><i>During interview:</i> Social Inattentiveness</p> <p>Distractibility/Poor Attention</p> <p>Uncooperativeness/Hostility</p> <p>Poor rapport</p> <p>Emotional Withdrawal (“invisible barrier”)</p>	

Confidence in assessment :
_____ 1 = Not at all – 5 = Very Confident

Explain here if validity of assessment is questionable :

- _____ Symptoms possibly drug-induced
- _____ Underreported due to lack of rapport
- _____ Underreported due to negative symptoms
- _____ Patient uncooperative
- _____ Difficulty to assess due to formal thought disorder
- _____ Other _____

Brief Psychiatric *Positive and Negative Syndrome* Rating Scale

Name/ID # : _____ Date: _____ Interviewer: _____

NA	1	2	3	4	5	6	7
Not Assessed	Not present	Very Mild	Mild	Moderate	Moderately Severe	Severe	Extremely Severe

Rate the following items on the basis of the patient's self-report during the interview (N.B. Items 7, G14, 12 and 13 are also rated on basis of observed behaviour)

1.	Somatic Concern (not nec. delusional)	1	2	3	4	5	6	7
G1	Somatic Concern	1	2	3	4	5	6	7
2./G2	Anxiety	1	2	3	4	5	6	7
3./G6	Depression	1	2	3	4	5	6	7
4.	Suicidality	1	2	3	4	5	6	7
5.	Guilt	1	2	3	4	5	6	7
G3	Guilt Feelings	1	2	3	4	5	6	7
6.	Hostility	1	2	3	4	5	6	7
G14	Poor Impulse Control	1	2	3	4	5	6	7
N4	Passive/Apathetic Social Withdrawal	1	2	3	4	5	6	7
7.	Elevated Mood	1	2	3	4	5	6	7
8.	Grandiosity	1	2	3	4	5	6	7
P5	Grandiosity	1	2	3	4	5	6	7
9.	Suspiciousness	1	2	3	4	5	6	7
P6	Suspiciousness/Persecution	1	2	3	4	5	6	7
G16	Active Social Avoidance	1	2	3	4	5	6	7
10./P3	Hallucinations	1	2	3	4	5	6	7
11./P1	Unusual Thought/ Delusions (Conviction, Preocc.,Imp.)	1	2	3	4	5	6	7
G9	Unusual Thought Content (Bizarreness)	1	2	3	4	5	6	7
12.	Bizarre Behavior	1	2	3	4	5	6	7
13.	Self-neglect	1	2	3	4	5	6	7
14./G10	Disorientation	1	2	3	4	5	6	7
N2	Emotional Withdrawal (from interpers. milieu)	1	2	3	4	5	6	7
N5.	Abstract thinking	1	2	3	4	5	6	7
P12.	Lack of Insight	1	2	3	4	5	6	7

Rate the following items on the basis of observed behaviour or speech of the patient during the interview.

15./P2	Concept. Disorganization	1	2	3	4	5	6	7
N6	Lack of Spontaneity & flow of Conversation	1	2	3	4	5	6	7
N7	Stereotyped Thinking	1	2	3	4	5	6	7
16./N1	Blunted Affect	1	2	3	4	5	6	7
17.	Emotional Withdrawal ("invisible barrier")	1	2	3	4	5	6	7
N3	Poor rapport (not psychosis-related)	1	2	3	4	5	6	7
P7	Hostility (during interview only)	1	2	3	4	5	6	7

G15	Preoccupation (absorption w. autistic exp.)	1	2	3	4	5	6	7
18./G7	Motor Retardation	1	2	3	4	5	6	7
19./G4	Tension	1	2	3	4	5	6	7
20.	Uncooperativeness(during interview only)	1	2	3	4	5	6	7
G8	Uncooperativeness (interpersonal non-compliance)	1	2	3	4	5	6	7
21./P4	Excitement	1	2	3	4	5	6	7
22./G11	Distractibility/Poor Attention	1	2	3	4	5	6	7
23.	Motor Hyperactivity	1	2	3	4	5	6	7
24./G5	Mannerisms and Posturing	1	2	3	4	5	6	7
G13	Disturbance of Volition	1	2	3	4	5	6	7

Confidence in assessment :
_____ 1 = Not at all – 5 = Very Confident

Explain here if validity of assessment is questionable :
_____ Symptoms possibly drug-induced
_____ Underreported due to lack of rapport
_____ Underreported due to negative symptoms
_____ Patient uncooperative
_____ Difficulty to assess due to formal thought disorder
_____ Other _____

Beck Depression Inventory

Name: _____ Marital Status: _____ Age: _____ Sex: _____
 Occupation: _____ Education: _____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness

- 0 I do not feel sad.
- 1 I feel sad much of the time.
- 2 I am sad all the time.
- 3 I am so sad or unhappy that I can't stand it.

2. Pessimism

- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to be.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

3. Past Failure

- 0 I do not feel like a failure.
- 1 I have failed more than I should have.
- 2 As I look back, I see a lot of failures.
- 3 I feel I am a total failure as a person.

4. Loss of Pleasure

- 0 I get as much pleasure as I ever did from the things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from the things I used to enjoy.
- 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

- 0 I don't feel particularly guilty.
- 1 I feel guilty over many things I have done or should have done.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

6. Punishment Feelings

- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

7. Self-Dislike

- 0 I feel the same about myself as ever.
- 1 I have lost confidence in myself.
- 2 I am disappointed in myself.
- 3 I dislike myself.

8. Self-Criticalness

- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all of my faults.
- 3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

10. Crying

- 0 I don't cry anymore than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying, but I can't.

11. Agitation

- 0 I am no more restless or wound up than usual.
- 1 I feel more restless or wound up than usual.
- 2 I am so restless or agitated that it's hard to stay still.
- 3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

- 0 I have not lost interest in other people or activities.
- 1 I am less interested in other people or things than before.
- 2 I have lost most of my interest in other people or things.
- 3 It's hard to get interested in anything.

13. Indecisiveness

- 0 I make decisions about as well as ever.
- 1 I find it more difficult to make decisions than usual.
- 2 I have much greater difficulty in making decisions than I used to.
- 3 I have trouble making any decisions.

14. Worthlessness

- 0 I do not feel I am worthless.
- 1 I don't consider myself as worthwhile and useful as I used to.
- 2 I feel more worthless as compared to other people.
- 3 I feel utterly worthless.

15. Loss of Energy

- 0 I have as much energy as ever.
- 1 I have less energy than I used to have.
- 2 I don't have enough energy to do very much.
- 3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

- 0 I have not experienced any change in my sleeping pattern.
- 1a I sleep somewhat more than usual.
- 1b I sleep somewhat less than usual.
- 2a I sleep a lot more than usual.
- 2b I sleep a lot less than usual.
- 3a I sleep most of the day.
- 3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

- 0 I am no more irritable than usual.
- 1 I am more irritable than usual.
- 2 I am much more irritable than usual.
- 3 I am irritable all the time.

18. Changes in Appetite

- 0 I have not experienced any change in my appetite.
- 1a My appetite is somewhat less than usual.
- 1b My appetite is somewhat greater than usual.
- 2a My appetite is much less than before.
- 2b My appetite is much greater than usual.
- 3a I have no appetite at all.
- 3b I crave food all the time.

19. Concentration Difficulty

- 0 I can concentrate as well as ever.
- 1 I can't concentrate as well as usual.
- 2 It's hard to keep my mind on anything for very long.
- 3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

- 0 I am no more tired or fatigued than usual.
- 1 I get more tired or fatigued more easily than usual.
- 2 I am too tired or fatigued to do a lot of the things I used to do.
- 3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

- 0 I have not noticed any recent change in my interest in sex.
- 1 I am less interested in sex than I used to be.
- 2 I am much less interested in sex now.
- 3 I have lost interest in sex completely.

Mania rating scales

Choose the one statement in each group that best describes the way you have been feeling for the past week including today. Circle the number next to the statement you picked.

Please note: the word "occasionally" when used here means once or twice; "often" means several times or more; "frequently" means most of the time.

<p>1)</p> <p>0 I do not feel happier or more cheerful than usual.</p> <p>1 I occasionally feel happier or more cheerful than usual.</p> <p>2 I often feel happier or more cheerful than usual.</p> <p>3 I feel happier or more cheerful than usual most of the time.</p> <p>4 I feel happier or more cheerful than usual all of the time.</p> <p>2)</p> <p>0 I do not feel more self-confident than usual.</p> <p>1 I occasionally feel more self-confident than usual.</p> <p>2 I often feel more self-confident than usual.</p> <p>3 I feel more self-confident than usual most of the time.</p> <p>4 I feel extremely self-confident all of the time.</p>	<p>3)</p> <p>0 I do not need less sleep than usual.</p> <p>1 I occasionally need less sleep than usual.</p> <p>2 I often need less sleep than usual.</p> <p>3 I frequently need less sleep than usual.</p> <p>4 I can go all day and night without any sleep and still not feel tired.</p> <p>4)</p> <p>0 I do not talk more than usual.</p> <p>1 I occasionally talk more than usual.</p> <p>2 I often talk more than usual.</p> <p>3 I frequently talk more than usual.</p> <p>4 I talk constantly and cannot be interrupted.</p> <p>5)</p> <p>0 I have not been more active (either socially, sexually, at work, home/ school) than usual.</p> <p>1 I have occasionally been more active than usual.</p> <p>2 I have often been more active than usual.</p> <p>3 I have frequently been more active than usual.</p> <p>4 I am constantly active or on the go all the time.</p>
--	---

Below are a number of statements describing how people sometimes feel. For each statement please indicate whether or not it applies to you. Simply circle YES if it applies to you, or NO if it does not apply to you. Please judge the statements on the basis of your feeling during the past week including today.

1. I can't sit still.	YES	NO
2. Lately I have been working much faster than usual.	YES	NO
3. I feel angry.	YES	NO
4. I have boundless energy.	YES	NO
5. I feel as though I can work 20 hours a day.	YES	NO
6. I feel like going on a spending spree.	YES	NO
7. I am constantly on the go.	YES	NO
8. People annoy me now more than before.	YES	NO
9. I move faster now than before.	YES	NO
10. Lately I feel like breaking things.	YES	NO
11. I've been telephoning a lot of friends recently.	YES	NO
12. I don't need as much sleep as other people.	YES	NO
13. I have been making new plans for travel.	YES	NO
14. I am continuously involved in activities.	YES	NO
15. I feel like being with people.	YES	NO
16. I make up my mind quickly.	YES	NO

Bipolar Longitudinal Investigation of Problems

Please think back on the last week and note if you have been aware of any of the following changes.

You would normally be expected to circle only one out of the “A” or “B” items in each section or just the “0” item, but please feel free to choose more than one item for each section, if applicable. For example, for Question 1 (ACTIVITY-PHYSICAL) you might have felt more active for some of the time but less active for the rest of the time, in which case you should circle the numbers next to the two items that most closely applied.

‘A’ Items

‘B’ Items

1. ACTIVITY – PHYSICAL

0 – I was no more or less active than usual and I think I appeared calm

- | | | | |
|----|---|----|--|
| A1 | I was slightly more active than usual. | B1 | I was slightly less active than usual. |
| A2 | I was moderately more active than usual. | B2 | I was moderately less active than usual. |
| A3 | I was considerably more active than usual. | B3 | I was considerably less active than usual. |
| A4 | I was excessively active and constantly driven to movement. | B4 | I was virtually inactive and felt exhausted. |

2. ACTIVITY – VERBAL

0 – I engaged in conversation and talked normally

- | | | | |
|----|--|----|--|
| A1 | I talked slightly more often and more rapidly in conversation. | B1 | I talked slightly less than usual in conversation. |
| A2 | I noticed a moderate increase in the rate and the amount I talked. | B2 | I noticed a moderate decrease in the rate and the amount I talked. |
| A3 | I felt very talkative. | B3 | I noted a considerable decrease in my speech. |
| A4 | I couldn’t stop myself talking. | B4 | I hardly said anything. |

3. THOUGHT PROCESSES

0 – My mind was alert and my speech coherent and easy to follow

- | | | | |
|----|--|----|--|
| A1 | My thoughts seemed to move a little faster. I sometimes deviated from my main topic of conversation. | B1 | I felt there was a slight slowing of my thoughts and my speech. |
| A2 | My thoughts were going fast and I regularly deviated from my main topic of conversation. | B2 | I felt a moderate slowing of my thoughts and speech. |
| A3 | My thoughts were going very fast and I often deviated from my main topic of conversation. | B3 | I noticed longer gaps between my sentences. |
| A4 | I was constantly distracted by the speed of my thoughts. | B4 | I had virtually nothing to say and my mind felt empty and blank. |

4. VOICE LEVEL

0 – The volume of my voice was the same as usual,
I spoke clearly and regulated my voice level according to the environment.

- | | | | |
|----|--|----|--|
| A1 | I spoke somewhat more loudly than usual. | B1 | I spoke somewhat more quietly than usual. |
| A2 | I spoke considerably more loudly than usual . | B2 | I spoke so quietly that others had to strain to hear. |
| A3 | I spoke very loudly, with little attention to the immediate environment. | B3 | I spoke very quietly, a lot more quietly than usual. |
| A4 | I felt like shouting and screaming and making lots of noise. | B4 | I volume of my voice was almost impossible for others to hear. |

5. **MOOD**

0 – The mood that I experienced was very much a normal, neutral mood.

- | | | | |
|----|--|----|---|
| A1 | My mood was a little better than usual. | B1 | My mood was slightly lower than usual. |
| A2 | I felt slightly elated. | B2 | I felt miserable. |
| A3 | I felt elated, emotionally high, joyful and exuberant. | B3 | I was really down and I often felt like crying. |
| A4 | I was so high that I was out of touch with my situation. | B4 | I was completely down and felt a sense of utter depression and gloom. |

6. **SELF-ESTEEM**

0 – My feelings of self-worth and esteem were the same as usual.

- | | | | |
|----|---|----|---|
| A1 | My self-esteem had slightly increased. | B1 | My self-esteem had slightly decreased. |
| A2 | My self-esteem had increased considerably. | B2 | My self-esteem had considerably decreased. |
| A3 | I felt that I was greatly admired and respected by other people and that I had particular talents or abilities. | B3 | My estimation of my own self-worth and abilities was extremely low. |
| A4 | I had grandiose ideas which I could not be dissuaded from. | B4 | I hated myself and felt worthless. My sense of self was so distorted that I could see no way to improve my situation. |

7. **CONTACT**

0 – I had normal emotional contact with others.

- | | | | |
|----|--|----|---|
| A1 | I felt sociable and tried to meet people. | B1 | I had a reduced wish or ability to be with people and was slightly withdrawn. |
| A2 | I chose to meet people but often became irritable. | B2 | I didn't feel like being with people. I had withdrawn to a moderate degree. |
| A3 | I felt very sociable and outgoing but behaved in a dominating way with others. | B3 | I felt isolated yet emotionally indifferent to others, even to friends and family, and I was withdrawing from everyone. |
| A4 | I constantly wanted to be with people but they did not like my dominating behaviour. | B4 | I felt totally isolated yet wanted no human contact. I was withdrawing from even the most important aspects of life. |

8. **SLEEP**

0 – My sleep was normal.

- | | | | |
|----|--|----|---|
| A1 | I took longer to fall asleep at night. | B1 | I had difficulty falling asleep at night. |
| A2 | I often woke up in the night but slept longer than usual. | B2 | I had difficulty falling asleep and woke up frequently. |
| A3 | I woke up before my usual time in the morning, but I slept longer overall and felt tired during the day. | B3 | My sleep was extremely disturbed having a marked effect on the way I felt during the day. |
| A4 | I wanted to sleep most of the time. | B4 | My inability to sleep properly was a major preoccupation and seemed to affect everything I did. |

9. **SEXUAL INTEREST**

0 – My level of sexual interest and activity was the same as usual.

- | | | | |
|----|--|----|--|
| A1 | There was a slight increase in my level of sexual interest and activity. | B1 | There was a slight decrease in my level of sexual interest and activity. |
| A2 | There was a moderate increase in my level of sexual interest and activity. | B2 | There was a moderate decrease in my level of sexual interest and activity. |
| A3 | There was a marked increase in my level of sexual interest and impulsiveness in sexual activity. | B3 | There was a marked decrease in my level of sexual interest and activity. |
| A4 | I was disinhibited and felt sexual constantly. | B4 | I had no interest whatsoever in sex and felt repulsed by the idea. |

10. EATING HABITS

0 – My eating habits were about the same as is usual for me, in amount, regularity and rate of eating.

- | | | | |
|----|--|----|--|
| A1 | My appetite was slightly decreased. I was eating slightly less than usual. | B1 | My appetite was slightly increased and I ate more faster and more often. |
| A2 | I felt moderately less desire for food than usual and ate more slowly. | B2 | I felt a moderately greater desire for food than usual and ate more quickly. |
| A3 | I had to force myself to eat my much reduced intake of food. | B3 | I was eating much more and more often than is usual for me. |
| A4 | I had no need for food at all and ate very little or nothing. | B4 | I was completely preoccupied by food with a marked increase in how much, how often and how fast I ate. |

11. WEIGHT CHANGE

0 – I weighed about the same as is usual for me.

- | | | | |
|----|--|----|---|
| A1 | I felt that I was losing weight, but not noticeable so. | B1 | I felt that I was putting on weight, but not noticeably so. |
| A2 | My weight was noticeably less than usual. | B2 | My weight was noticeably more than usual. |
| A3 | I had lost a great deal of weight. | B3 | I had put on a great deal of weight. |
| A4 | My weight was very noticeably lower than was usual for me. I was worried about the amount of weight I had lost | B4 | My weight was very noticeably greater than is usual for me. I was worried about the amount of weight that I had put on. |

12. MEANING

0 – I had not noticed any change in what I regard as important in my life.

- | | | | |
|----|--|----|--|
| A1 | I had noticed certain everyday things meant more to me. | B1 | I had some doubts about what is meaningful in my life. |
| A2 | I had noticed more significance in certain things than I would normally. | B2 | Questions of meaning and purpose were of concern to me. |
| A3 | I had become aware of great significance becoming attached to things/events/people. | B3 | I was frequently concerned that there was no meaning or purpose to life. |
| A4 | I felt that I had a more profound understanding and/or awareness and I was convinced of my pivotal role in the scheme of things. | B4 | My life felt completely without meaning or purpose. |

13. ANXIETY

0 – I felt no more or less anxious, insecure or tense than usual.

- | | | | |
|----|---|----|---|
| A1 | I felt more anxious than usual. | B1 | I felt less anxious than usual. |
| A2 | I felt I was in a state of anxiety which was difficult to control and which interfered with my daily life. | B2 | I felt much less anxious than usual. |
| A3 | My feelings of anxiety and experience of inner unrest, nervousness and panic often interfered with my daily life. | B3 | I felt positively more relaxed and worried about things a lot less. |
| A4 | Feelings of panic were present so often that They constantly interfered with my daily life. | B4 | I felt remarkably calm and tranquil. |

14. FEELINGS OF PRESSURE

0 – I felt that there was no more or less pressure on me than usual.

A1	I was putting myself under slightly less pressure than usual.	B1	I was putting myself under slightly more pressure than usual.
A2	I did not feel under pressure.	B2	I felt under pressure.
A3	I felt detached from my responsibilities.	B3	I felt a strong sense of responsibility and that it was important that I try to satisfy and respond to all the pressures and demands upon me.
A4	I had no feeling of pressure at all and felt completely separate and detached from any sense of responsibility.	B4	I had to keep going though under the weight of enormous pressure.

15. PASSAGE OF TIME

0 – My perception of the passing of time was no different than usual.

A1	Time seemed to be passing slower than usual than usual.	B1	Time seemed to be passing more quickly than usual.
A2	I felt unable to fill all of my time constructively.	B2	I was fully occupied and I feared I would not fit everything in that I wanted to.
A3	Time dragged so much and passed so slowly that I dreaded the future.	B3	Time was passing so quickly that I felt pressured to keep up.
A4	I felt almost as if time had stopped and that the future would not happen.	B4	Time was flying by so fast that I didn't have time to stop and think.

16. FUTURE PLANS

0 – My ability to plan for the future was the same as usual for me.

A1	I was slightly more able than usual to make plans for the future.	B1	I occasionally questioned whether I should plan for the future.
A2	I was thinking of more future plans than usual.	B2	I sometimes thought there was no point in planning for the future.
A3	My head was full of many plans and ideas.	B3	I frequently thought there was no point in planning for the future.
A4	I couldn't stop myself from constantly thinking of unrealistic plans for the future.	B4	The future seemed completely hopeless to me.

17. PAIN SENSITIVITY

0 – My sensitivity to pain was the same as usual for me.

A1	I felt slightly less sensitive to pain than usual.	B1	I felt slightly more sensitive to pain than usual.
A2	I definitely felt less sensitive to pain than usual.	B2	I definitely felt more sensitive to pain than usual.
A3	My capacity to tolerate pain felt very high.	B3	My capacity to tolerate pain felt extremely low.
A4	I felt incapable of experiencing pain ever again.	B4	I felt completely overrun with pain.

18. WORK

0 – My capacity for work was the same as usual for me.

A1	I had a slightly increased capacity for work.	B1	I had a slightly decreased capacity for work.
A2	I had a greatly increased capacity for work.	B2	I had a greatly decreased capacity for work.
A3	My capacity for work was extremely high.	B3	My capacity for work was extremely low.
A4	I felt I had an infinite capacity for work and could take on anything.	B4	I had no capacity for work whatsoever and the slightest demand overwhelmed me.

INDIVIDUAL CHARACTERISTICS

Please feel free to personalise any of the stated items if you think they are relevant to you but in a slightly different way, or add additional items at the end of the questionnaire if there are other items relevant for you that have not been mentioned at all.

1. GENERAL HEALTH

- 0 I had no particular health worries.
- 1 I had been worrying more about my health.
- 2 I had frequent worries that I had symptoms I could not explain.
- 3 I was increasingly preoccupied by thoughts of the serious illnesses I might have.
- 4 I was convinced that I had a life threatening illness.

2. SMOKING

- 0 I did not smoke/I had stopped smoking.
- 1 I had started smoking again/I continued to smoke as often as usual.
- 2 I smoked a slightly increased number per day e.g. 10-15 extra.
- 3 I smoked a greatly increased number per day e.g. a packet or more, chain smoking.
- 4 I smoked constantly.

3. ALCOHOL

- 0 I drank the same amount as was normal for me.
- 1 I drank slightly more or slightly more often than usual.
- 2 There was a moderate increase in the amount or frequency of my intake.
- 3 There was a marked increase in my alcohol consumption.
- 4 I was intoxicated most of the time and often incapacitated.

4. HOSTILITY/DESTRUCTIVENESS

- 0 I felt no more or less irritable than usual.
- 1 I felt slightly more irritable than usual, when provoked.
- 2 I felt angrier and more aggressive than usual and felt more likely to lose my temper.
- 3 I felt argumentative, lost my temper and become more aggressive without provocation but the resulting conflict could be resolved.
- 4 I felt unable to control my feelings of violent anger and destructiveness.

5. SPENDING

- 0 I was spending money in the same amounts and at the same rate as usual.
- 1 I was spending slightly more money than usual, not excessively so.
- 2 I was spending more freely and impulsively than usual.
- 3 I was spending money indiscriminately and inappropriately.
- 4 I was grossly overspending, way over my budget.

6. RECREATIONAL DRUG HABITS

- 0 I continued my abstinence/my normal use.
- 1 My use was slightly increased compared with normal.
- 2 My use was moderately increased compared with normal.
- 3 My use was markedly increased compared with normal.
- 4 My use of recreational drugs had become excessive and extreme.

7. RELIGIOSITY (this item refers to feelings described as, e.g. religious, spiritual, mystical, significant, meaningful).

- 0 My customary practice and strength of feelings were as is usual for me.
- 1 I was more aware than usual of this aspect of my life, in my thoughts.
- 2 I actively participated more and felt more strongly in my spiritual life.
- 3 I felt drawn to and preoccupied by strong feelings, which seemed most meaningful.
- 4 I was completely preoccupied by a profound, personal spiritual experience.

8. RESPONSE TO FRUSTRATION

- 0. I dealt with frustration in a calm and ordered way.
- 1. I disliked it when others prevented me from getting my way.
- 2. Frustration of my efforts upset me and I reacted accordingly.
- 3. I reacted with a short fuse to frustration of my plans.
- 4. I felt violently angry when others tried to restrict me.

9. MEDICATION

- 0. I continued with my usual scheme of prescribed medication.
- 1. I occasionally altered my dose of medication (increasing or decreasing).
- 2. I deliberately changed the dose I took, against medical instructions.
- 3. I stopped taking my prescribed medication i.e. active discontinuation.
- 4. My symptoms returned after discontinuation and I required medication to return to a stable state.

10. INCONSIDERATE ACTIVITY

- 0. I was aware of the feelings of others to the same degree as usual for me.
- 1. I adjusted my behaviour according to the needs of others less than usual.
- 2. I felt disinhibited and put my own wants ahead of those of others.
- 3. I did not care what effect my behaviour had on others.
- 4. My behaviour was constantly upsetting others but I still did not care at all.

11. CLOTHING

- 0. My style of dress was the same as usual for me.
- 1. I was more aware of my choice and style of dress than usual.
- 2. I felt like wearing louder, more colourful clothing than usual.
- 3. I wanted to look way out and be unusual in my style of dress.
- 4. I imbued certain items and colours with specific meaning.

12. CREATIVITY

- 0. I felt no more or less creative than is usual for me.
- 1. My appreciation was slightly enhanced of things e.g. music, colour, literature.
- 2. I was coming up with more new ideas than is usual.
- 3. I felt drawn to spend much of my time doing creative things e.g. writing, art.
- 4. I was preoccupied by spontaneous ideas and I had to follow my urges to be creative, e.g. to make something, paint, play an instrument, write.

13. DAILY VARIATION IN MOOD

- 0. My moods were not generally associated with time of day.
- 1. I noticed slight mood changes according to time of day.
- 2. My mood was predictable by time of day.
- 3. I experienced very powerful swings in mood according to time of day.
- 4. Time of day completely determined my mood.

14. CONCENTRATION

- 0. There was no change in my ability to concentrate.
- 1. I needed to make an effort to concentrate.
- 2. Even with effort, it was difficult to concentrate.
- 3. My lack of concentration affected my memory and decision making.
- 4. I found it virtually impossible to concentrate e.g. I couldn't follow a conversation or TV programme.

15. IMPULSIVITY

- 0. I noticed no unusual or abnormal impulses.
- 1. I felt more impulsive but this was controllable.
- 2. I noticed the urge to act on impulses more strongly.
- 3. I found it increasingly difficult to resist acting on impulses.
- 4. I felt I had no control over my impulses whatsoever.

16. SUICIDAL IMPULSES

- 0. I had experienced no impulses to harm myself.
- 1. I had occasional thoughts of harming myself or wanting to be unconscious to get away from things.
- 2. I had frequent wishes to be dead and had reflected on how I could achieve this by my own actions.
- 3. I had thought out how I would kill myself and the plan came to mind regularly.
- 4. I had a constant urge to end my life and I was preoccupied by plans for achieving this.

17. MENSTRUAL CYCLE (WOMEN)

- 0. I did not associate my menstrual cycle with any marked mood swings.
- 1. I noticed slight premenstrual mood swings.
- 2. I expected to experience mood changes before the start of my period.
- 3. My marked premenstrual mood swings eased once the period stopped.
- 4. I experienced prolonged, marked and extreme mood changes premenstrually which persisted during the period and afterwards.

18. SEASONALITY

- 0. My mood was unaffected by changing seasons.
- 1. I experienced minor mood changes in relation to season.
- 2. I experienced significant mood changes in relation to season.
- 3. I experienced major mood changes in relation to season.
- 4. I experienced extreme mood changes in relation to season.

19. JET LAG

- 0. My mood was unaffected by jet lag
- 1. I noticed a slight change in my mood because of jet lag.
- 2. My mood showed a definite change because of jet lag.
- 3. I noticed a considerable change in my mood because of jet lag.
- 4. My mood changed dramatically because of jet lag.

PLEASE NOTE ANY OTHER CHANGES THAT YOU ARE AWARE OF BUT WHICH HAVE NOT BEEN MENTIONED ELSEWHERE IN THE QUESTIONNAIRE:

1.....

2.....

3.....

Quality of Life

This part of the questionnaire asks how you feel about your quality of life, health, and other areas of your life. Please answer all the questions. If you are unsure about which response to give to a question, please choose the one that appears most appropriate. This can often be your first response. Please keep in mind your standards, hopes, pleasures and concerns. We ask that you think about your life in the last two weeks. For example, thinking about the last two weeks, a question might ask:

	Not at all	Little	Moderate amount	Very much	Extreme Amount
How much do you worry about your health	1	2	3	④	5

You should circle the number that best fits how much you have worried about your heath over the last two weeks. So you would circle the number 4 if you were worried about your health “Very much” as above.

	Not at all	Little	Moderate amount	Very much	Extreme Amount
How much do you worry about your health	①	2	3	4	5

You should circle the number 1 if you have worried “Not at all” about your health. Please read each question, assess your feelings, and circle the number on the scale for each question that fives the best answer for you.

QUESTIONS

1.	Very Good	Good	Fair	Poor
In general, would you say your health is:	1	2	3	4

2.	Very Poor	Poor	Neither Poor nor good	Good	Very Good
How would you rate your quality of life?	1	2	3	4	5

3.	Very Dissatisfied	Dissatisfied	Neither satisfied nor dissatisfied	Satisfied	Very satisfied
How satisfied are you with your health?:	1	2	3	4	5

The following questions ask about **How Much** you have experienced certain things in the last two weeks:

4.	Not at all	Little	Moderate amount	Very much	Extremely
To what extent do you feel that (physical) pain prevents you from doing what you Need to do?	1	2	3	4	5
How much do you need any medical treatment to function in your daily life?	1	2	3	4	5
How much do you enjoy life?	1	2	3	4	5
To what extent do you feel your life to be meaningful?	1	2	3	4	5

5.	Not at all	Little	Moderate amount	Very much	Extremely
How well are you able to concentrate?	1	2	3	4	5
How safe do you feel in your daily life?	1	2	3	4	5
How healthy is your physical environment?	1	2	3	4	5

The following questions ask about **How Completely** you experienced or were able to do certain things in the last two weeks:

6.	Not at all	Little	Moderate	Mostly	Completely
Do you have enough energy for everyday life?	1	2	3	4	5
Are you able to accept your bodily appearance?	1	2	3	4	5
Have you enough money to meet your needs?	1	2	3	4	5
How available to you is the information that you need in your day to day life?	1	2	3	4	5
To what extent do you have the opportunity for leisure activities?	1	2	3	4	5

The following questions ask you to rate how **Satisfied, Happy Or Good** you have felt about various aspects of your life over the last two weeks:

7.	Very dissatisfied	Dissatisfied	Neither satisfied nor dissatisfied	Satisfied	Very satisfied
How satisfied are you with your sleep?	1	2	3	4	5
How satisfied are you with your ability to perform your daily living activities?	1	2	3	4	5
How satisfied are you with your capacity for work?	1	2	3	4	5
How satisfied are you with yourself?	1	2	3	4	5
How satisfied are you with your personal relationships?	1	2	3	4	5
How satisfied are you with your sex life?	1	2	3	4	5
How satisfied are you with the support you get from your friends?	1	2	3	4	5
How satisfied are you with the conditions of your living place?	1	2	3	4	5
How satisfied are you with your access to health services?	1	2	3	4	5
How satisfied are you with your transport?	1	2	3	4	5

8.	Very poor	Poor	Neither poor nor good	Good	Very good
How well are you able to get around?	1	2	3	4	5

The following question refers to **How Often** you have felt or experienced certain things, for example the support of your family or friends or negative experiences such as feeling unsafe.

9.	Never	Seldom	Quite Often	Very Often	Good
How often do you have negative feelings such as blue mood, despair, anxiety, depression?	1	2	3	4	5

Personal Beliefs about Illness Questionnaire

Please indicate whether you strongly disagree, disagree, agree or strongly agree by ticking the appropriate box.

	<i>Strongly Disagree</i>	<i>Disagree</i>	<i>Agree</i>	<i>Strongly Agree</i>
1. I will always need to be cared for by professional medical staff.				
2. My illness frightens me.				
3. I am embarrassed by my illness.				
4. I am capable of very little as a result of my illness.				
5. Because of my illness I have to rely on psychiatric services.				
6. There must always have been something wrong with me to have caused my illness.				
7. I find it difficult to cope with my current symptoms.				
8. My illness is too delicate/brittle for me to work or keep a job.				
9. I know when I'm relapsing but I can't do anything about it.				
10. My illness is a judgement on me.				
11. I am powerless to influence or control my illness.				
12. I am fundamentally normal; my illness is like any other.				
13. Society needs to keep people with my illness apart from everyone else.				
14. There must be something about my personality that causes me to be what I am.				
15. I can talk to most people about my illness.				
16. There is something strange about me that causes my illness.				

Meta Cognitions Questionnaire

This questionnaire is concerned with beliefs people have about their thinking. Listed below are a number of beliefs that people have expressed. Please read each item and say how much you generally agree with it by circling the appropriate number. Please respond to all the items, there are no right or wrong answers.

	<i>Do not agree</i>	<i>Agree slightly</i>	<i>Agree moderately</i>	<i>Agree very much</i>
1. Worrying helps me to avoid problems in the future.	1	2	3	4
2. My worrying is dangerous for me.	1	2	3	4
3. I have difficulty knowing if I have actually done something, or just imagined it.	1	2	3	4
4. I think a lot about my thoughts.	1	2	3	4
5. I could make myself sick with worrying.	1	2	3	4
6. I am aware of the way my mind works when I am thinking through a problem.	1	2	3	4
7. If I did not control a worrying thought, and then it happened, it would be my fault.	1	2	3	4
8. If I let my worrying thoughts get out of control, they will end up controlling me.	1	2	3	4
9. I need to worry in order to remain organised.	1	2	3	4
10. I have little confidence in my memory for words and names.	1	2	3	4
11. My worrying thoughts persist, no matter how I try to stop them.	1	2	3	4
12. Worrying helps me to get things sorted out in my mind.	1	2	3	4
13. I cannot ignore my worrying thoughts.	1	2	3	4
14. I monitor my thoughts.	1	2	3	4
15. I should be in control of my thoughts all the time.	1	2	3	4
16. My memory can mislead me at times.	1	2	3	4
17. I could be punished for not having certain thoughts.	1	2	3	4
18. My worrying could make me go mad.	1	2	3	4
19. If I do not stop my worrying thoughts, they could come true.	1	2	3	4
20. I rarely question my thoughts.	1	2	3	4
21. Worrying puts my body under a lot of stress.	1	2	3	4
22. Worrying helps me to avoid disastrous situations.	1	2	3	4
23. I am constantly aware of my thinking.	1	2	3	4
24. I have a poor memory.	1	2	3	4
25. I pay close attention to the way my mind works.	1	2	3	4
26. People who do not worry, have no depth.	1	2	3	4
27. Worrying helps me cope.	1	2	3	4

	<i>Do not agree</i>	<i>Agree slightly</i>	<i>Agree moderately</i>	<i>Agree very much</i>
28. I imagine having not done things and then doubt my memory for doing them.	1	2	3	4
29. Not being able to control my thoughts is a sign of weakness.	1	2	3	4
30. If I did not worry, I would make more mistakes.	1	2	3	4
31. I find it difficult to control my thoughts.	1	2	3	4
32. Worrying is a sign of a good person.	1	2	3	4
33. Worrying thoughts enter my head against my will.	1	2	3	4
34. If I could not control my thoughts I would go crazy.	1	2	3	4
35. I will lose out in life if I do not worry.	1	2	3	4
36. When I start worrying, I cannot stop.	1	2	3	4
37. Some thoughts will always need to be controlled.	1	2	3	4
38. I need to worry, in order to get things done.	1	2	3	4
39. I will be punished for not controlling certain thoughts.	1	2	3	4
40. My thoughts interfere with my concentration.	1	2	3	4
41. It is alright to let my thoughts roam free.	1	2	3	4
42. I worry about my thoughts.	1	2	3	4
43. I am easily distracted.	1	2	3	4
44. My worrying thoughts are not productive.	1	2	3	4
45. Worry can stop me from seeing a situation clearly.	1	2	3	4
46. Worrying helps me to solve problems.	1	2	3	4
47. I have little confidence in my memory for places.	1	2	3	4
48. My worrying thoughts are uncontrollable.	1	2	3	4
49. It is bad to think certain thoughts.	1	2	3	4
50. If I do not control my thoughts, I may end up embarrassing myself.	1	2	3	4
51. I do not trust my memory	1	2	3	4
52. I do my clearest thinking when I am worrying.	1	2	3	4
53. My worrying thoughts appear automatically.	1	2	3	4
54. I would be selfish if I never worried.	1	2	3	4
55. If I could not control my thoughts, I would not be able to function.	1	2	3	4
56. I need to worry, in order to work well.	1	2	3	4
57. I have little confidence in my memory for actions.	1	2	3	4
58. I have difficulty keeping my mind focused on one thing for a long time.	1	2	3	4

	<i>Do not agree</i>	<i>Agree slightly</i>	<i>Agree moderately</i>	<i>Agree very much</i>
59. If a bad thing happens which I have not worried about, I feel responsible.	1	2	3	4
60. It would not be normal, if I did not worry.	1	2	3	4
61. I constantly examine my thoughts.	1	2	3	4
62. If I stopped worrying, I would become glib, arrogant and offensive.	1	2	3	4
63. Worrying helps me to plan the future more effectively.	1	2	3	4
64. I would be a stronger person if I could worry less.	1	2	3	4
65. I would be stupid and complacent not to worry.	1	2	3	4
66. I must control my thoughts or I will become ill.	1	2	3	4
67. It's dangerous for me to think about my thoughts.	1	2	3	4
68. When I start to feel good, I might become unwell.	1	2	3	4
69. I constantly examine the way I feel.	1	2	3	4
70. My thoughts are harmful to me.	1	2	3	4
71. I have to have control over my emotions.	1	2	3	4
72. My thoughts are interesting.	1	2	3	4
73. I cannot influence what I think.	1	2	3	4
74. My emotions are harmful to me.	1	2	3	4
75. My thoughts are going too fast.	1	2	3	4
76. I cannot follow what I think.	1	2	3	4
77. My thoughts are confusing.	1	2	3	4
78. I have to be careful how I feel.	1	2	3	4
79. Only I am aware of what I think.	1	2	3	4
80. I need to be careful what I think.	1	2	3	4
81. I try and avoid thinking about my thoughts.	1	2	3	4
82. I can become ill if I don't control the way I feel.	1	2	3	4
83. I don't trust my thoughts.	1	2	3	4
84. My thoughts are unreliable.	1	2	3	4
85. Other people know what I am thinking.	1	2	3	4
86. If I could control my feelings there would be something wrong with me.	1	2	3	4
87. My thoughts control me.	1	2	3	4
88. I cannot tell people about my thoughts.	1	2	3	4
89. I am embarrassed by my thoughts.	1	2	3	4
90. I control my thoughts.	1	2	3	4
91. I cannot trust my feelings.	1	2	3	4

Please ensure that you have responded to all items. Thank you.

Dysfunctional Attitudes Scale

This scale lists different attitudes or beliefs which people sometimes hold.

Please read each statement carefully and decide how much you agree or disagree with what it says.

For each of the attitudes, please indicate your answer by placing a tick (4) under the column that **best describes how you think**. Be sure to choose only one answer for each attitude. But please note that because people are different, there is no 'right' or 'wrong' answer to these statements.

To decide whether a given attitude is typical of your way of looking at things, simply keep in mind what you are like **most of the time**.

Attitudes	Totally Agree	Agree Very Much	Agree Slightly	Neutral	Disagree Slightly	Disagree Very Much	Totally Disagree
1. If I fail partly, it is as bad as being a complete failure.							
2. If others dislike you, you cannot be happy.							
3. I should be happy all the time.							
4. People will probably think less of me if I make a mistake.							
5. My happiness depends more on other people than it does on me.							
6. I should always have complete control over my feelings.							
7. My life is wasted unless I am a success.							
8. What other people think about me is very important.							
9. I ought to be able to solve my problems quickly and without a great deal of effort.							
10. If I don't set the highest standards for myself, I am likely to end up a second rate person.							
11. I am nothing if a person I love doesn't love me.							

Attitudes	Totally Agree	Agree Very Much	Agree Slightly	Neutral	Disagree Slightly	Disagree Very Much	Totally Disagree
12. A person should be able to control what happens to him/her.							
13. If I am to be a worthwhile person, I must be truly outstanding in at least one major respect.							
14. If you don't have other people to lean on, you are bound to be sad.							
15. It is possible for a person to be scolded and not get upset.							
16. I must be a useful, productive, creative person or life has no purpose.							
17. I can find happiness without being loved by another person.							
18. A person should do well at everything he/she undertakes.							
19. If I do not do well all the time, people will not respect me.							
20. I do not need the approval of other people in order to be happy.							
21. If I try hard enough, I should be able to excel at anything I attempt.							
22. People who have good ideas are more worthy than those who do not.							
23. A person doesn't need to be well liked in order to be happy.							
24. Whenever I take a chance or risk I am only looking for trouble.							

Rosenberg self esteem scale

This is a short questionnaire to measure thoughts about yourself. Please indicate whether you strongly disagree, disagree, agree, or strongly agree with each statement.

	<i>Strongly disagree</i>	<i>Disagree</i>	<i>Agree</i>	<i>Strongly agree</i>
1. On the whole I am satisfied with myself.				
2. At times I think I am no good at all.				
3. I feel I have a number of good qualities.				
4. I am able to do things as well as most other people.				
5. I feel I do not have much to be proud of.				
6. I certainly feel useless at times.				
7. I feel I am a person of worth, at least equal to others.				
8. I wish I could have more respect for myself.				
9. All in all, I am inclined to feel I am a failure.				
10. I take a positive attitude towards myself.				

Significant Others Scale

For each person listed below please circle a number from 1 to 7 to show how well he or she provides the type of help that is listed.
The second part of each question asks you to rate how you would like things to be if they were exactly as you hoped for. As before please put a circle round one number between 1 and 7 to show what your rating is.

Person 1 – Partner

		Never	Sometimes			Always		
1	a) Can you trust, talk to frankly and share your feelings with this person?....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
2	a) Can you lean on and turn to this person in times of difficulty?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
3	a) Does he/ she give you practical help?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
4	a) Can you spend time with him/ her socially?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
5	a) Can you get physical comfort from him/ her?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7

Person 2 – A Close Relative (state relationship.....).

		Never	Sometimes			Always		
1	a) Can you trust, talk to frankly and share your feelings with this person?....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
2	a) Can you lean on and turn to this person in times of difficulty?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
3	a) Does he/ she give you practical help?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
4	a) Can you spend time with him/ her socially?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
5	a) Can you get physical comfort from him/ her?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7

Person 3 – A Close Friend

		Never	Sometimes			Always		
1	a) Can you trust, talk to frankly and share your feelings with this person?....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
2	a) Can you lean on and turn to this person in times of difficulty?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
3	a) Does he/ she give you practical help?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
4	a) Can you spend time with him/ her socially?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7
5	a) Can you get physical comfort from him/ her?.....	1	2	3	4	5	6	7
	b) What rating would your ideal be?.....	1	2	3	4	5	6	7

Family Emotional Involvement and Criticism Scale

Please describe your family. Below are several statements with numbers from 1 to 5; please circle one number beside every statement, describing how appropriate the statement is.

1

2

3

4

5

Almost never

Once in a While

Sometimes

Often

Almost Always

*Almost
Never*

Sometimes

*Almost
Always*

1) I am upset if anyone else in my family is upset.

1 2 3 4 5

2) My family is critical of me.

1 2 3 4 5

3) My family knows what I am feeling most of the time.

1 2 3 4 5

4) My family approves of most everything I do.

1 2 3 4 5

5) My family members don't intrude in my life.

1 2 3 4 5

6) My family finds fault with my friends.

1 2 3 4 5

7) My family members try to make decisions for me.

1 2 3 4 5

8) My family complains about the way I handle money.

1 2 3 4 5

9) I do things with my family members more often than I do things with my friends.

1 2 3 4 5

10) My family approves of my friends.

1 2 3 4 5

11) Family members give me money when I need it.

1 2 3 4 5

12) My family complains about what I do for fun.

1 2 3 4 5

13) My family knows what I am thinking before I tell them.

1 2 3 4 5

14) My family is always trying to change me.

1 2 3 4 5

15) It is difficult to get time alone in my family.

1 2 3 4 5

16) I have to be careful what I do or my family will put me down.

1 2 3 4 5

17) I often know what my family members are thinking before they tell me.

1 2 3 4 5

18) I find myself frequently judging my family.

1 2 3 4 5

19) If I am upset, people in my family get upset too.

1 2 3 4 5

20) I wish my family members would be more like me.

1 2 3 4 5

21) My family has no idea what I am feeling most of the time.

1 2 3 4 5

22) My family members are too involved in my life.

1 2 3 4 5

23) If I have no way of getting somewhere my family will take me.

1 2 3 4 5

Life Events Inventory

Please indicate whether any of the following events have happened to you in the past six months by marking the appropriate box to indicate whether an event had a good or bad effect overall on your life.

Good Bad

- | Good | Bad | |
|------------------------------|--------------------------|--|
| 1. <input type="checkbox"/> | <input type="checkbox"/> | You had a serious physical illness or injury |
| 2. <input type="checkbox"/> | <input type="checkbox"/> | A close relative had a serious illness (from which they did not die) |
| 3. <input type="checkbox"/> | <input type="checkbox"/> | You have been pregnant (or partner) |
| 4. <input type="checkbox"/> | <input type="checkbox"/> | You had a miscarriage |
| 5. <input type="checkbox"/> | <input type="checkbox"/> | You had an abortion |
| 6. <input type="checkbox"/> | <input type="checkbox"/> | You experienced sexual difficulties |
| 7. <input type="checkbox"/> | <input type="checkbox"/> | Your spouse or partner died |
| 8. <input type="checkbox"/> | <input type="checkbox"/> | An immediate family member died |
| 9. <input type="checkbox"/> | <input type="checkbox"/> | A close friend or relative died |
| 10. <input type="checkbox"/> | <input type="checkbox"/> | You gained a new family member (immediate) |
| 11. <input type="checkbox"/> | <input type="checkbox"/> | You got married |
| 12. <input type="checkbox"/> | <input type="checkbox"/> | There has been an increase in serious arguments with your spouse or partner |
| 13. <input type="checkbox"/> | <input type="checkbox"/> | There has been an increase in arguments with other family members (e.g. children) |
| 14. <input type="checkbox"/> | <input type="checkbox"/> | There have been serious problems with a close friend, neighbour or relative not living at home |
| 15. <input type="checkbox"/> | <input type="checkbox"/> | Your son or daughter left home |
| 16. <input type="checkbox"/> | <input type="checkbox"/> | Your children went into care of others |
| 17. <input type="checkbox"/> | <input type="checkbox"/> | You experienced trouble or behaviour problems with your children |
| 18. <input type="checkbox"/> | <input type="checkbox"/> | Your spouse or partner began or stopped working |
| 19. <input type="checkbox"/> | <input type="checkbox"/> | You have separated from your spouse or partner |
| 20. <input type="checkbox"/> | <input type="checkbox"/> | You have reconciled with your spouse or partner |
| 21. <input type="checkbox"/> | <input type="checkbox"/> | You began an extramarital affair |
| 22. <input type="checkbox"/> | <input type="checkbox"/> | You experienced a break up of an affair |
| 23. <input type="checkbox"/> | <input type="checkbox"/> | Your spouse/ partner began an extramarital affair |
| 24. <input type="checkbox"/> | <input type="checkbox"/> | You have divorced or ended a steady relationship |
| 25. <input type="checkbox"/> | <input type="checkbox"/> | A new person came to live in your household (apart from a new baby) |
| 26. <input type="checkbox"/> | <input type="checkbox"/> | You studied for, or did, important exams |
| 27. <input type="checkbox"/> | <input type="checkbox"/> | You failed an important exam |
| 28. <input type="checkbox"/> | <input type="checkbox"/> | You have been unemployed or seeking work for a month or more |
| 29. <input type="checkbox"/> | <input type="checkbox"/> | You began to have trouble or disagreements with your supervisors at work |
| 30. <input type="checkbox"/> | <input type="checkbox"/> | You had a big change in the hours or conditions of your work |
| 31. <input type="checkbox"/> | <input type="checkbox"/> | You started in a completely different type of job |
| 32. <input type="checkbox"/> | <input type="checkbox"/> | Your income increased substantially (25%) |
| 33. <input type="checkbox"/> | <input type="checkbox"/> | Your income decreased substantially (25%) |
| 34. <input type="checkbox"/> | <input type="checkbox"/> | You had debts you were unable to repay |
| 35. <input type="checkbox"/> | <input type="checkbox"/> | You went on holiday |
| 36. <input type="checkbox"/> | <input type="checkbox"/> | You moved house |
| 37. <input type="checkbox"/> | <input type="checkbox"/> | You purchased your own house (taking out mortgage) |
| 38. <input type="checkbox"/> | <input type="checkbox"/> | You got new neighbours |
| 39. <input type="checkbox"/> | <input type="checkbox"/> | You had quarrels with neighbours |
| 40. <input type="checkbox"/> | <input type="checkbox"/> | You had minor difficulties with the police or the authorities (speeding, drunkenness) |
| 41. <input type="checkbox"/> | <input type="checkbox"/> | You were sentenced to jail |
| 42. <input type="checkbox"/> | <input type="checkbox"/> | You were involved in a fight |
| 43. <input type="checkbox"/> | <input type="checkbox"/> | An immediate family member started drinking heavily |
| 44. <input type="checkbox"/> | <input type="checkbox"/> | An immediate family member attempted suicide |
| 45. <input type="checkbox"/> | <input type="checkbox"/> | An immediate family member was sent to prison |
| 46. <input type="checkbox"/> | <input type="checkbox"/> | You had problems relating to alcohol and drugs |
| 47. <input type="checkbox"/> | <input type="checkbox"/> | You experienced serious restrictions of your social life |
| 48. <input type="checkbox"/> | <input type="checkbox"/> | You experienced a period of homelessness (e.g. hostel or sleeping rough) |
| 49. <input type="checkbox"/> | <input type="checkbox"/> | Something you valued or cared for greatly was stolen or lost |
| 50. <input type="checkbox"/> | <input type="checkbox"/> | Other Events (please specify): |

Please indicate whether any of these events has happened to you in the past five years.

If you can remember, please indicate the year when the event happened.

Good Bad Year

- | | | | |
|--------------------------|--------------------------|-------|--|
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you had a serious illness or injury? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Has a close relative had a serious illness or injury? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you or your partner had a miscarriage or an abortion? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Has anyone close to you, family or friend died? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you broken off a steady relationship? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you had any serious problems or major arguments with a close friend, neighbour or relative? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you had to give up a training course or educational course that was important to you? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you failed any important exams? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you been forced to leave a job for any reason? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you been unemployed for a month or more? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you had debts you were unable to pay? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you been attacked, raped or assaulted? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you been burgled or had your property stolen or damaged? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you had any involvement with the police, the courts or the legal profession? |

If you have ever been married or lived with a partner in the last five years: -

- | | | | |
|--------------------------|--------------------------|-------|---|
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you separated from your partner for a month or more? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you had a legal separation? |
| <input type="checkbox"/> | <input type="checkbox"/> | _____ | Have you been divorced or begun divorce procedures? |

Follow up interview guide

FOLLOW-UP 6/ 18 MONTHS AFTER THERAPY

Date: ____/____/____

Initials: _____

Subject No.: _____

Date of birth ____/____/____

Age: _____

Gender: _____

Mood (rate from -10 to +10) = _____

RELAPSE SECTION

MOOD

1. Have you experience any major mood changes? Yes/No

If “Yes”,

2. How many **depressions**?

3. When did your last episode start?(date)

4. How long did it last?(in days)

5. To what extent did this episode disrupt your daily life?

6. How many **manic** episodes?

7. When did your last manic episode start?(date)

8. How long did it last?(in days)

9. To what extent did this episode disrupt your daily life?

HOSPITAL

10. Were you admitted to hospital for either mania or depression? Yes/No

If “Yes”,

11. How many times since the end of therapy?

12. How long on each occasion?

SUBSYNDROMAL MOOD SWINGS

13. Have you experienced any minor mood changes? (Describe)

MEDICATION

14. Are you currently on any medication? Yes/No

If “Yes”,

15. What medications are you currently being prescribed? (HOW MUCH?)

Medication	Quantity
1.	
2.	
3.	

16. Has your medication changed since your last therapy session? Yes/No.....if “Yes,

17. What was changed? (PREVIOUS DOSE, CURRENT DOSE) and When?

Medication	Previous dose	Current dose	When?	Is this a decrease?	
				YES	NO

CONTACT WITH PROFESSIONALS

18. Do you have contact with any professionals? Yes/No.....If "Yes",

CODING

1= every wk, 2= every 2 wks, 3 = every 4wks, 4 = every 6wks, 5 = every 8wks, 6 = every 12wks or less

Professional	Who?	(How often DID you see him/her?)	How often DO you NOW see him/her?	Has the amount of contact decreased? (tick box)	
				YES	NO
G.P.					
Psychiatrist					
CPN					
Social Worker					
Other					

19. How satisfied are you with this usual/changed arrangement? RATE 1-5 (1= very dissatisfied; 5=satisfied)

LIFE EVENTS

20. Have you experienced any LIFE EVENTS since the therapy ended? Yes/No

If "Yes",

Life Events since the end of therapy	Good or bad (or both) effect on your life?
1.	
2.	
3.	
4.	
5.	

Total no. of bad life events =

Total no. of good life events =

COPING STRATEGIES

21. What strategies have you used to cope with these life events/things in general?

COPING STRATEGIES
1. Cognitive strategies:
2. Behavioural strategies:
3. EWS
4. Interpersonal strategies:

22. Which of the above strategies did you utilise most?

23. Which of the above strategies were most effective?

RELATIONSHIPS/social support

24. How would you describe your current relationships? (Generally)

Relatives:

25. Excluding the people that live with you, how many relatives live within 20 miles?

26. With how many of these are you in contact with? (either face to face, or non face to face i.e. telephone, email, letter)

27. How often? (state how many times per week/month or year) (elicit this information if possible)

28. Have any of these relationships significantly changed?

If "Yes",

29. How many have improved/been positive changes?

30. How many have been negative changes?

Friends:

31. Excluding the people that live with you, how many friends live within 20 miles?

32. With how many of these are you in contact with? (either face to face, or non face to face i.e. telephone, email, letter)

33. How often? (state how many times per week/month or year) (elicit this information if possible)

34. Have any of these relationships significantly changed?

If "Yes",

35. How many have improved/been positive changes?

36. How many have been negative changes?

WORK/STUDY/ROLE

37. Are you working at present?

(CODING 0=N/a; 1=Unemp; 2=Emp; 3=student/education; 4=self emp)

If "Yes",

38. Are you working full time or part time?

39. Is this a change since the end of therapy? Yes/No

If "Yes",

40. How satisfied are you with this change?

RATE 1-5 (1= very dissatisfied; 5=satisfied)

THERAPY

41. What did you find HELPFUL/UNHELPFUL about therapy? (RANK IN TERMS OF IMPORTANCE below:)

RANK	Helpful things:	Comments
	Un-helpful things:	

42. WHAT WOULD THEY WANT TO BE DIFFERENT ABOUT THERAPY?
(rank in terms of most important = 1 etc).

-
-
-

43. Were there any CRITICAL POINTS/TURNING POINTS IN THE THERAPY?
If "Yes",

44. What were the critical points? (and why)

45. FEEDBACK ON THE THERAPISTS

46. FURTHER PSYCHOLOGICAL SUPPORT? (Yes/No)

47. PERMISSION TO FOLLOW UP AT A LATER DATE? (Yes/No)

48. ANY OTHER COMMENTS?

49. Feedback on the patients (group patients only)

50. Cohesion of the group?

(5 = very good, 4 = good, 3 = o.k., 2 = not very good, 1 = very poor)

51. Recreational drug use/alcohol use/any other mood altering behaviour

What Drug?	How much?	How often?